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THE HEART IN HYPERTHYROIDISM

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THE great importance of cardiac involvement in hyperthyroidism is practically unappreciated in this country. Most of the text-books and systems of medicine pass it by with scant comment, and some show an almost unbelievable state of ignorance on the subject.

In order better to understand cardiac involvement in hyperthyroidism, all the cases of that disease, numbering 141, in the records of the Medical Service of the Peter Bent Brigham Hospital to the year 1922 inclusive, were examined; and an extensive survey of the literature was undertaken for purposes of comparison.

Caleb Hiller Parry,[1] the old Bath physician to whom the credit for the first clear description of hyperthyroidism Osler said should be given, included his eight cases of the disease, seen from 1786 on, under the section of "Diseases of the Heart." He devoted fully ten pages to clear and systematic description of a peculiar disease of the heart attended with exophthalmos and thyroid enlargement. Flajani,[2] by whose name the disease is sometimes known, also recorded, in 1802, heart disturbances in a disease characterized by a tumor in the anterior part of the neck. Adelmann,[3] in 1828, was the first definitely to associate goitre with certain types of heart disease, and coined the word *Kropfherz* or goitre-heart. Von Basedow,[4] after whom the disease is chiefly known, knew of heart disturbances in a disease characterized by goitre, cardiovascular symptoms, and exophthalmos.

The great group of Dublin physicians of the early nineteenth century was well aware of the relationship existing between heart disease and enlargement of the thyroid gland and protuberant eyeballs. Sir Henry Marsh,[5] in 1841, described two cases of enlargement of the thyroid, exophthalmos, and heart disease. Irregularity of the heart was noted in the first case, with dilatation of its cavities. MacDonnel[6] also gives two cases of "a peculiar form of disease of the heart, attended with enlargement of the thyroid gland and eyeballs," and says that Graves and Stokes are the only two who know anything about this disease. Graves[7] gives clear descriptions of the affection, while Stokes,[8] in his book on "Diseases of the Heart and the Aorta," states that the point of departure of the disease is the heart, after which come the peculiar symptoms of goitre, exophthalmos, etc.

During the middle of the nineteenth century, several articles were written on the simultaneous appearance of goitre, exophthalmos, and cardiac disturbances. Egeberg[9] in 1850 described a case of heart-failure in a woman of 34 years with struma and exophthalmos. The disease in which exophthalmos, goitre, and heart disturbances were found came to be known as an entity, and its symptoms were described by Lubarsch,[10] and by Nathanson[11] in 1850. Naumann[12] in 1852 described a case of this entity which had severe heart pain and a greatly enlarged heart. Taylor,[13] in the same year, was the first American author to write on the disease.

At a meeting of the Boston Society for Medical Improvement, held in 1858,[14] reported in the BOSTON MEDICAL AND SURGICAL JOURNAL, a symposium on "anaemic exophthalmos" was held. Bigelow reported a case, and showed a daguerreotype of the patient. Morland described a case ending evidently in heart-failure and death, while Jones' case showed cardiac irregularity and "a loud bellows-murmur after the first sound of the heart."

Prael,[15] a Berlin ophthalmologist, in 1857 pointed out the necessity of looking for heart symptoms in exophthalmos and goitre, as in only two cases of nine with which he was familiar were there no heart symptoms. In the same year, Markham[16] of London cited a case in which autopsy showed marked cardiac changes.

It was only in 1877, however, that the great importance of cardiac disturbances in goitre was emphasized, when Rose[17] read before the German Surgical Congress his paper on "Goitre Death and the Radical Operation for Goitre." To one American physician, Lockbridge,[18] the importance of heart symptoms was so evident that he proposed naming the disease, "cardiac exophthalmic goitre."

Charcot,[19] writing in the latter part of the nineteenth century, pointed out the fact that heart affections frequently appeared in patients in whom one or more of the signs of the disease were lacking, and he named this condition *"formes frustes"* of Basedow's disease. The conception that the cases of hyperthyroidism with cardiac involvement represent an incomplete form of the disease has been handed down by French authors and is still persistent.

For some time after Rose's article, the profession seemed to settle down with the knowledge that cardiovascular symptoms were part of the picture and required no further study. Except for a scattered and unimportant article here and there, there was nothing in the literature specifically relating to the clinical importance of the heart in hyperthyroidism until Kraus[20] in 1899 first introduced the conception of the toxic goitre heart. This occasioned great interest both abroad and in this country, but it is only from 1906, when a symposium on thyroid disease was held in the course of a medical convention in Berlin, that the modern period for cardiac study in Graves' disease may be said to have begun.

Since then, several excellent articles have appeared, and a few monographs have been written: by Moutet,[21] Minnich,[22] Ter-Poghossian,[23] and Iszlondski;[24] while excellent accounts relating to the heart appear in monographs on hyperthyroidism or the thyroid by Kocher,[25] Sattler,[26] and Boothby.[27] Kocher, Murray,[28] Mosse,[29] and Monnier[30] were the first to publish any extensive analyses of cases on the condition of the heart in the disease, but exact studies were not made until the last few years; these were by Krumbhaar,[31] White and Aub[32] and in 1922 by Hamilton,[33] and Kerr and Hensel.[34]

Even by recent writers, the paramount importance of the cardiac condition in the disease, aside merely from its theoretical interest, has not been at all emphasized. Of all the present writers, Goodall[35] alone seems to be the one who has not missed the point (or what I believe to be the point) in regard to the importance of these disturbances.

THEORIES

Although the fact that heart symptoms are of common and constant occurrence in hyperthyroidism, the mechanism of their production is not at all clear. Various theories have naturally been proposed from time to time, which I will briefly review. They may be classed under two headings, the mechanical and the toxic.

Rose,[17] in 1877, first gave the mechanical theory as the cause of the heart symptoms. He maintained that tracheal stenosis, due to an enlarged thyroid gland, changed the pressure relations in the thorax, so that there was an overfilling of the right heart during inspiration, with later secondary dilatation and hypertrophy. Kocher[25] thought that the goitre heart was due to congestion of the large veins by continued pressure of the thyroid. Wölfler[36] stressed the hindered expiration due to enlarged thyroid, causing indirectly strain on the right heart and degeneration of the myocardium. Another mechanical theory (quoted by Bauer[37]) was that the continued pressure of the thyroid on the nerves to the heart caused its degeneration.

Increased metabolism, by causing the heart to work more, and then gradually wearing it out, is the accepted theory of many writers. This view is well put forth by Boas,[38] in a recent paper.

The toxic theory was first put forth by Kraus in 1899, and since then the mechanical theory has become gradually weaker and weaker until it is now practically extinct. Assuming that the cardiac symptoms are due to a toxemia, investigators have found great difficulty in determining the nature of the toxin, and the mode of its action. De Cyon[39] and Minnich[22] maintained that the thyroid is degenerated in goitrous conditions, and that the products of degeneration affect the cardiac nerves. Bircher[40] also thought that the thyroid underwent degeneration, but that the products affected the myocardium directly. Kraus[20] and Lubarsch[10] thought that the thyrotoxic goitre hearts were dependent upon a quantitatively changed secretion—an overfunction of the thyroid. On the other hand, Cleret and Gley,[41] Lampe and Liesegang[42] and others were just as emphatic that the condition was dependent upon a qualitatively changed secretion—a dysfunction of the thyroid. McCarrison[43] thinks that hyperthyroidism, with all its symptoms, is a direct result of intestinal toxemia. Bigler,[44] after a lengthy discussion, comes to the conclusion that the heart symptoms are probably of toxic origin, source obscure.

At the present time, the mechanical theory is practically discarded, while the conception of a toxemia or toxicosis has rapidly replaced it in favor.

THE NORMAL HYPERTHYROID HEART

In the 141 cases of hyperthyroidism, 75, or 53.2 per cent. of the hearts, were considered normal. The criteria set for a normal heart were that its size be within normal limits, its valves without murmurs except a blowing systolic, its electrocardiogram normal in every respect, and its function good—as shown by the absence of symptoms of heart-failure. To the absolutely normal hearts may be added those in which was found a minor abnormality by electrocardiogram (16, or 11.3 per cent.), and those which showed some hypertrophy but no clinical signs of heart damage (4, or 2.8 per cent.). The total number of hearts essentially normal is thus 95, or 67.4 per cent. The cases which showed hypertrophy and nothing else were somewhat of a puzzle, as it was a problem whether to put them into the group of normal or of damaged hearts. They were finally placed into the group of essentially normal, since they were physiologically so.

It is difficult to compare these figures with those of other authors, for many have considered even tachycardia as one of the criteria of heart damage. Hamilton found about 90 per cent. of his cases normal, while Bigler considered about 90 per cent. of his to be damaged. Various figures in between these two have been given.

What are the characteristics of the heart which is normal for the disease hyperthyroidism? They may be discussed under the headings of subjective and objective symptoms.

Of the subjective symptoms, *palpitation* is the most common. Brought on by the slightest mental or physical action, or constantly present, it is no wonder that this sensation of strong pounding in the chest should unnerve the already nervous patient and cause her to complain of palpitation above all else. Attacks of palpitation are frequently paroxysmal; if they occur at intervals of a week or more, especially if accompanied by precordial distress, a paroxysmal irregularity should be strongly suspected. Indeed, Kerr and Hensel believe that all attacks of palpitation probably represent attacks of paroxysmal auricular fibrillation or flutter.

An important subjective symptom is *cardiac pain.* Authors of the present generation seem to belittle this symptom. Goodall and Kerr and Hensel say that true anginal attacks are a rare occurrence in hyperthyroidism, and are joined by Renner,[45] who says that hyperthyroid patients have no great precordial pain. Most authors do not even mention attacks of pain occurring during the disease.

In the older literature, references to angina pectoris occurring in the course of exophthalmic goitre are more common. Parry's fifth case showed marked precordial pain, as did one of MacDonnel's two cases.

Naumann[12] in 1853 reported a case with enlargement of the thyroid and exophthalmos which died after typical anginal attacks; autopsy showed myocardial changes but no coronary blocking. Trousseau,[46] in his several articles on the disease, mentions cases of angina pectoris occurring during its course. Pierre Marie[47] observed a typical case of angina in a 21-year-old male. Several authors, quoted by Sattler, described typical cases of angina pectoris in Basedow's disease. One of six cases reported by Symmers[48] in 1918 showed precordial pain.

Precordial pain or distress was recorded in 19 cases, or 13.5 per cent., of this series. Of these, 10 cases occurred in patients with essentially normal hearts, while 9 occurred in damaged heart cases. In other words, 10.5 per cent. of the normal hearts, 22.5 per cent. of the damaged hearts had pain.

The pain may simply be in the nature of a distress, a feeling of fullness in the chest; this is more common in the cases without heart damage. Those with damage, if they have pain, have the typical attack of angina pectoris, with the sweating, premonition of death, etc. It is noteworthy, however, that even in the undamaged cases, occurring at ages as early as 21, precordial pain, indistinguishable from the so-called typical attack of angina, may occur. This symptom occurring in a young woman without evident heart damage should make one wonder as to hyperthyroidism. Inquiries as to whether the pulse was irregular during the attack may occasionally give one valuable information.

The objective symptoms of the normal heart in hyperthyroidism are quite striking. The *apex impulse* is often described in the records as diffuse, and can be seen over three or four intercostal spaces. The heart pounds against the hand, giving it a distinct shock. Some clinicians estimate roughly the extent of the hyperthyroidism by the amount of shock the heart gives to the hand. Due probably to this shock, a *thrill* is often noted, and was described in 43, or 30.5 per cent., of the cases. The thrill is described as either presystolic or systolic (the timing being often a matter of individual variation), and is usually placed at the apex.

Tachycardia is, of course, the outstanding symptom. Cases showing it over a long period usually represent a hidden goitre. The rate varies tremendously from day to day, even from hour to hour, in the same patient, going easily from 90 to 130 and down again. If the rate jumps to 160 or over, irregularities should be looked for and, if possible, an electrocardiogram taken.

The heart sounds are extremely loud, especially the first. Due probably to lengthening and loudness of the first sound, a *presystolic murmur* may be described by some observers, especially if they are attempting to pin the tag "*mitral stenosis*" to the case. A loud, blowing *systolic murmur,* heard usually over the entire precordia, is the rule in hyperthyroidism, only 10 or 15 of the entire series failing to show it. The pulmonic second sound is usually louder than normal.

The vascular symptoms are interesting. There seems to be great lability in the control of the vasomotor system. The patient blushes freely and spasmodically. A capillary pulse is often described.

Coincident with, and probably as a result of the increased force of the heartbeat, the peripheral vessels pulsate markedly. The carotids are especially affected, the pulsations being so striking as to be recorded in about a quarter of the cases. A whistling sound, a bruit, is heard over the carotids and their branches, particularly over the thyroid arteries. A "pistol-shot," similar to the one heard in aortic regurgitation, may often be heard over the brachials and femorals. The abdominal aorta is often seen to pulsate, and pulsations of the liver and spleen have been observed (Kocher,[25] Gerhardt[49]). Pulsations, slight movement, and widening of the retinal arteries in their beds were first observed by Becher[50] in 1873.

The blood-pressure in hyperthyroidism has long been a subject of much discussion. Many authors agree that the pressure is lowered; others that it is not abnormal; others that it is very variable, and still others that it is usually increased.

Falta,[51] Dalmady,[52] Chvostek,[53] Leidner,[54] Bigler,[44] and Goodall[35] maintain that the blood-pressure is usually low, or on the low border of normal. Goodall goes quite deeply into the question of blood-pressure, and concludes that the systolic pressure in the typical case is low, and that the pulse pressure is also low, but that there are primary and secondary changes in the cardiovascular system.

Spiethoff,[55] Donath,[56] Kraus[20] and others emphasize the great variability of readings, not only in different cases, but in the same patients from day to day.

Kraus, Brown,[57] Elliott,[58] Plummer,[59] Taussig,[60] Barr,[61] Federn,[62] Krumbhaar,[31] White,[32] Kerr and Hensel,[34] Boothby,[27] and others say that the blood-pressure is usually high, and that there is in most cases a high pulse pressure.

In this series of cases, only 10, or 7.1 per cent., showed a blood-pressure lower than 120 mm. systolic, and of these only four were below 110, none below 100; the lowest was 104, so that really there were no hypotensive cases.

Among the cases with normal hearts, the average blood-pressure was 140 mm. systolic, 69.5 mm. diastolic; while among the damaged heart cases it was 152 mm./75.5 mm., or a grand average of 146 mm./72.5 mm. The pulse pressure is seen to be on the average very high, and this agrees well with the feel of the pulse, which is very much akin to the Corrigan. The average of the damaged hearts is seen to be rather considerably higher than that of the normal; in fact, the damaged hearts show a hypertension.

Twenty-one of the normal cases showed a hypertension, that is, a systolic pressure above 150; of these, six were above 180 mm.; 15 of the damaged cases showed a hypertension, and of these, 3 were above 180.

No attempt has been made to correlate the amount of the pulse pressure with the pulse rate and the metabolism. Boothby says a good index to the metabolism is the combined knowledge of the pulse rate and the pulse pressure. Read[63] states that there is close correlation between the basal metabolism and the other two. He concludes that the basal pulse rate and pressure may be used as a rough clinical guide to the basal metabolism.

Forceful action—these two words well sum up the characteristics of the heart in hyperthyroidism. Forceful action produces a diffuse, pounding impulse, thrill, loud sounds, pulsations of vessels, sounds over vessels, and high systolic pressure.

Before leaving the subject of the heart normal for hyperthyroidism, it might be well to review the electrocardiographic findings. Practically all authors, among them Krumbhaar, Leidner,[54] Stein,[64] Hoffman,[65] Strubel,[66] and Kerr and Hensel note the frequent occurrence of high T waves, the most constant of electrocardiographic findings in hyperthyroidism. Both Krumbhaar and Leidner have indicated the prognostic value of the T wave. Usually high, when it is flattened or inverted in Leads I and II, it indicates a bad prognosis. This is also the conclusion of Kerr and Hensel. Diagnostically, Leidner states that a high T wave is a constant finding and may be a differential point in distinguishing a case of hyperthyroidism from a neurosis. Goodall found large P waves characteristic, and Kerr and Hensel say that the P waves may be widened.

In this series of cases, five EKG'S showed T waves prominent enough to excite comment in the records; but there were several others with high T waves not so noted. No careful study of the prognostic value of a low T wave was made, but I would agree with the conclusions of Krumbhaar and Leidner on this point. Left ventricular hypertrophy or preponderance was noted frequently.

DAMAGED HEARTS

Since very few extensive analyses of cases of hyperthyroidism for cardiac involvement have been made, the statistics in regard to cardiac damage are scanty. In general, it may safely be said that 25 per cent. would probably represent the average figure, taking into account the fact that some authors have considered even tachycardia a symptom of cardiac damage (which it may well be), while others have, short of actual heart failure, considered all cases normal.

I have attempted to take a mid-ground position between these two extreme views. What I have considered a normal heart, has already been pointed out. The criteria of what constitutes a damaged heart, I have grouped into three classes, and a case, in order to be classed as damaged, need fall into one of the following three groups:

1. Cases with an irregularity (aside from sinus arrhythmia or extrasystoles alone), without heart failure: 12 cases.
2. Cases with signs of heart-block: 5 cases.
3. Cases with signs of heart-failure: 23 cases.

Forty cases, or 28.5 per cent., were thus considered to have damaged hearts.

Cases with myocarditis due to some known cause, such as chronic valvular disease, chronic nephritis, etc., were excluded as far as possible. Doubtful cases were also excluded.

1. *Cases with Arrhythmia.*

From Parry on, arrhythmia has been considered a frequent symptom, or one which appeared with such frequency as to warrant notice by practically all authors who have ever written on the subject. Pierre Marie[47] found one arrhythmic case in eleven exophthalmics, Griffith[67] two in 26, Kocher[25] 4 in 97, Murray[28] 12 in 180, Schultze[68] 2 in 50, Mosse[29] 1 in 128, Bigler[44] 40 in 100, and Hewlett[69] 5 in 15. It is noteworthy that none of these authors described the arrhythmia or mentioned its type. It is only within the past few years that detailed statements as to the type of irregularity, their exact description, and their classification have been made, and these practically entirely by American authors.

Krumbhaar,[31] in an electrocardiographic study, found three cases of auricular fibrillation, one transient, and one case of auricular flutter among 51 cases of hyperthyroidism. White,[32] in 47 cases, found three with paroxysmal auricular fibrillation, three with permanent auricular fibrillation, and one with paroxysmal auricular flutter. About 10 per cent. of Hamilton's[33] 200 cases showed auricular fibrillation, paroxysmal or persistent. Kerr and Hensel,[34] in 58 cases, found 20 with auricular fibrillation, 6 with flutter; 11 of these were paroxysmal in type. Willius[70] estimates that 9 per cent. of exophthalmic goitre and 70 per cent. of toxic thyroid adenoma show auricular fibrillation.

In this series of cases there were 22, or 15.6 per cent., with arrhythmias as follows:

Cases with Arrhythmia

10—Auricular fibrillation.
9—Paroxysmal auricular fibrillation.
1—Paroxysmal auricular flutter.
1—Paroxysmal auricular fibrillation and flutter.
1—Paroxysmal auricular tachycardia.

Cases of complete heart-block, although usually irregular, were not classed among the irregular hearts.

It has seemed to me wise to make a distinction between the ordinary permanent irregularities and those which were paroxysmal. The cases showing permanent auricular fibrillation or flutter all showed signs of heart-failure, and were therefore classed among that group. On the other hand, none of the cases showing paroxysmal auricular arrhythmia showed (while they were paroxysmal) symptoms of heart-failure.

Paroxysmal Arrhythmia.

Although some of the earlier writers mentioned transient arrhythmia as occurring occasionally in their cases, and a few of the recent authors speak of their frequency, their importance has not yet been realized. Money[71] in 1886 reported a case whose heart rate suddenly went to 200 and remained there for 20 minutes. Hingston Fox[72] in 1891 took pulse tracings of a case which showed periods of transient irregularity every few days or week. Gerhardt[49] in 1893 reported a case with paroxysms of irregularity and extremely rapid rate; v. Hösslin[73] and Kobisch[74] also reported such cases in 1896. His[75] observed intermittent irregularities in one of his cases, as did Sattler.[26] Chvostek[53] notes that fleeting irregularities may appear during the course of the disease. Huber[76] observed two cases of paroxysmal irregularity. Levine,[77] in observing 18 cases of paroxysmal auricular fibrillation, found one in a case of hyperthyroidism. The observations of Krumbhaar, White and Aub, Hamilton, and Kerr and Hensel have already been noted.

In this series, 12 cases with paroxysmal irregularities were observed. Of these, nine were paroxysmal auricular fibrillation, one flutter, one fibrillation and flutter, and the twelfth paroxysmal auricular tachycardia.

A short résumé of these cases may be of interest and value.

Case 5315: Female, 36 years. Typical hyperthyroid symptoms. Severe precordial pain in attacks. Metabolism +57. Heart negative except that a peculiar regular irregularity was observed. Electrocardiograms taken showed at times normal curves, at times auricular flutter. Precordial pain found associated with attacks of flutter.

Case 6444, 6587, 9031: Male, 37 years. Typical hyperthyroid symptoms. Attacks of severe precordial pain, associated with attacks of paroxysmal auricular fibrillation, shown clinically and by EKG*. Metabolism +70. A year after, fibrillation was no longer paroxysmal but permanent.

Case 8402, 14009, 15678: Female, 37 years. Six years before entrance the patient had been operated upon for exophthalmic goitre. On entry her heart at first seemed normal. A month later EKG showed paroxysmal auricular flutter, which recurred at intervals. Two years later, her metabolism was +74, her heart was now enlarged, and there was still paroxysmal auricular fibrillation. On her last entry, a year later, she showed permanent auricular fibrillation and flutter, and she died in a year.

Case 10819: Female, 22 years. Metabolism +70. Heart negative except for attacks of paroxysmal auricular tachycardia associated with palpitation. She died shortly post-operative.

Case 13404: Female, 55 years. Hyperthyroid symptoms, metabolism +29, as well as severe precordial pain. Heart negative except that there were occasional lapses to auricular fibrillation, associated with precordial pain.

Case 17177: Male, 33 years. Metabolism +85. Heart somewhat enlarged, rate usually 100, occasionally very high with irregular rhythm. Attacks of paroxysmal auricular fibrillation were shown by EKG and these were associated with attacks of palpitation.

Case 18932: Female, 44 years. Complaint chiefly of attacks of dyspnea. The patient was considered a heart case since she showed few hyperthyroid signs, which were only brought out by subsequent examinations. Metabolism +55 to +69. Auricular fibrillation at entry, normal rhythm for three weeks, then a sudden attack of cardiac distress and dyspnea heralded auricular fibrillation, demonstrated by EKG.

Case 19377: Female, 49 years. Hyperthyroid symptoms, metabolism +70 to +95. Heart was considered normal until three weeks after entrance when very rapid and irregular heart action was discovered, proven to be auricular fibrillation by EKG. This was the only time it occurred during the patient's stay.

Case 18837, 18975: Male, 47 years. Metabolism +66. Heart was considered normal until a surgeon, who observed the patient immediately after some exertion (against advice), noticed the very rapid, irregular pulse with marked pulse deficit. This was noted several times later, the rapid pulse rate being associated with the paroxysmal fibrillation.

*Electrocardiogram.

Case 17966, 19066, 19338: Female, 54 years. Complaint of palpitation, nausea, vomiting, dyspnea, and some edema. No exophthalmos or thyroid enlargement. This was considered a heart case, until, on the suggestion of one of the visiting physicians, a metabolism done showed +39. EKG's at various intervals showed paroxysmal auricular fibrillation, as well as partial heart block (idio-ventricular beats), and left ventricular preponderance.

Case 17999, 18872: Female, 32 years. Metabolism +45. Heart negative. Patient was operated upon; one day post-operative, the heart action became quite irregular for long periods, with constant bigeminy and occasional trigeminy.

Case 18955, 19412: Female, 42 years. Metabolism +54. Heart considered normal on first entry, in July, 1922. Two days after re-entry in September, 1922, paroxysmal auricular fibrillation was noted and shown by EKG. This was the only arrhythmic period.

Several lessons may be learned from the above series of cases. First, as to *age*. One case was 22 years old, five were between 30 and 40, four between 40 and 50, one 54, and the last 55. The average age for this group is 39.5 years, while that for the so-called heart-failure group is 52.3 years, or about 12 years higher.

Second, as to the co-existence of *subjective symptoms* with the attacks of irregularities: In three of the above 12 cases, the occurrence of severe *precordial pain* was recorded. *Palpitation,* coming in attacks, is very frequent. Kerr and Hensel state that the occurrence of definite attacks of palpitation in hyperthyroid patients should lead one to suspect paroxysmal irregularities. Palpitation as such was recorded in only two of the above cases, but I am sure they occurred in attacks in all. Acute attacks of dyspnea occurred in one case.

The third and very interesting observation to be gleaned from these cases is the occurrence of permanent irregularity, coupled of course with heart-failure, *provided the case with paroxysmal irregularity was allowed to go on unoperated.* This was shown in three cases: 6444, 8402, and 17177.

The fourth observation is that occasionally a paroxysmal irregularity may appear after operation, and only then. This was shown in Case 17999.

2. *Cases with Heart-Block.*

The occurrence of heart-block in hyperthyroidism, whether complete or partial, passes almost unnoticed in the literature. I could find but six references. Two of these, by Elliott[58] and Sloan,[78] merely mention block *en passant.* Merklen[79] publishes the report of a case, in 1880, evidently of the acute onset of a block in a goitrous patient. The only reports that are of value are by Goodall, Krumbhaar, and Kerr and Hensel. Goodall states that prolongation of the p-r interval is a sign of a severe, long-standing case, with the myocardium definitely damaged. Krumbhaar observed delayed conduction time in 2 of 51 cases. Kerr and Hensel found delayed conduction time in 3 of 25 cases studied by electrocardiograph.

In this series, there were five cases which showed some degree of block. These, with short descriptions, are the following:

Case 4075: Female, 36 years, with a goitre of ten years' standing, showed an enlarged regular heart; EKG showed premature auricular and nodal beats, and delayed conduction time. Metabolism +60.

Case 15812: Female, 31 years. Typical hyperthyroid symptoms. First heart sound not heard. Protodiastolic gallop rhythm at apex. EKG showed greatly delayed a-v conduction time.

Case 6223, 8032, 8300: Male, 29 years. Although there was a partial thyroidectomy a few years previously, the patient had a metabolism of +60 on his first entry, heart apparently normal. A year later, his heart was enlarged, irregular, and there was *complete heart-block.* Later EKG's showed partial (2:1) block, and then delayed conduction time, finally normal tracings.

Case 11823: Male, 26 years. Typical hyperthyroid symptoms, metabolism +65. Heart apparently normal, with normal EKG tracings. After five months in bed without operation, the patient developed persistent severe precordial pain. The next day, the pulse was slow and irregular. EKG showed an irregular and atypical type of heart-block, with an irregular idioventricular rhythm. Diagnosis was probable *complete heart-block* with an irregular idioventricular rhythm. The metabolism rose to +99 and then to +120. The next month EKG showed normal tracings, but two weeks later there was 3:2 block. The patient was sent home unoperated.

Case 3414: Female, 60 years. Hyperthyroid symptoms; metabolism not determined. Heart apparently normal. EKG showed defective conduction in the right branch of the bundle of His.

Except for the last case, which is of a type different from the others, the cases of block all occurred in young people—36, 31, 29, 26 years.

The presence of delayed conduction time is interesting in that it has recently been taken to mean acute myocardial damage.

Most interesting are the two cases of complete heart-block coming on under observation, and in apparently normal hearts. The first case (6223) was entirely paroxysmal, and cleared up by passing through stages of partial block. The second case was an unfortunate one of a young man who, after five months in bed, developed complete block.

It is noteworthy that the metabolism, wherever recorded in this group, was of the highest, +60 or more.

3. *Cases with Heart-Failure.*

The occurrence of heart-failure in the course of hyperthyroidism has been noted by practically all authors who have ever written on the subject. Indeed, as I mentioned before, hyperthyroidism was at one time considered by some as an unusual and peculiar form of heart disease, attended by exophthalmos, goitre, etc.

In this series, there were 23 cases which showed signs of heart-failure only, cases of paroxysmal arrhythmia and of heart-block being excluded.

Auricular fibrillation (permanent), considered as a symptom of heart-failure, occurred in 10 of these 23 cases.

As these cases are studied, one is impressed by the presence of two distinct types. The first type is that of the ordinary full-blown case of hyperthyroidism occurring usually in a person about 40 or under, with all the classical symptoms of exophthalmic goitre, including a high metabolism. A case report of this type follows:

Case 18974: Female, 43. P. H. negative. Two years ago, patient noticed a goitre, then all the symptoms of hyperthyroidism,—palpitation, exophthalmos, tremor, loss of weight, etc.,—developed. On examination, she showed moderate exophthalmos, symmetrically enlarged thyroid, tremor, etc. The heart was not enlarged to percussion; the heart rate varied from 90 to 150, average 130. The sounds and rhythm were absolutely irregular, auricular fibrillation being confirmed by EKG. A systolic blow was present. The B.P. was 188/82, and the metabolism +45.

The second type appears to me to be the most interesting, since its occurrence has been recognized by very few in this country, and by only a few more abroad. In this type, the patient is older, and in almost all cases a woman. She has but few hyperthyroid symptoms, by no means the full array presented in the first type. There may be merely tremor and goitre, plus cardiac symptoms. Exophthalmos is not usually present, but there may be a staring expression to the eyes. The patient is hardly ever emaciated, and her skin is often pigmented. On close questioning, it may be learned that she has had a small goitre for years, twenty or more, and has often had attacks of palpitation and heart pain. She may even remark that at certain periods in the year she has felt more excited than usually, has been unable to do the housework, has lost weight. The chief complaint on entrance is usually with reference to the heart—dyspnea, edema, etc.—and only on careful questioning and observation are the atypical hyperthyroid symptoms brought out, whereupon, if the metabolism be determined, it is shown to be elevated. The metabolism in these cases, unlike that of the first type, is usually between +15 and +30, hardly ever above +40. A case report of this type follows:

Case 19945: Female, 55 years. The patient complained of a sense of painful oppression over her chest at the level of the midsternum, and of frequent attacks of sharp pain below the left breast. These symptoms had been present for some years. Palpitation was of one year's duration. On examination, the heart was naturally considered to be the organ at fault. It was enlarged,—15-16 cm. out from the midline,—rate 128; a systolic blow was present. B.P 155/70. Vital capacity 75 per cent. of normal. It was noted by one observer that the thyroid gland was slightly and diffusely enlarged; by another that the isthmus was palpable, and that the right lobe seemed to contain a lobule. There was no exophthalmos, but a fine tremor of the hands was present.

Because of the general resemblance of this case to others that had turned out to be cases of hyperthyroidism, a metabolism determination was done and shown to be +49.

This type of case has been well described by Boothby[27] in The Oxford Medicine under the heading of adenomatous or colloid goitre existing for years, without exophthalmos, and with long-continued, slightly increased metabolic rate. Charcot[19] called these cases *"formes frustes"* of exophthalmic goitre, in which, with mild hyperthyroid symptoms, there are marked cardiac signs. Kraus,[20] in 1906, called attention to these cases in which the cardiopathy was progressive, the thyroid symptoms being mild, and attributed them to the effect of toxin from a more or less diseased thyroid. His,[75] in the same year, proposed calling this type *"cardiopathia thyreogenes levis,"* a highly descriptive but quite unwieldy name. Gittermann,[80] in 1907, studied this problem from the opposite point of view, by observing the cases of myogenic disease of the heart in a goitre region (Nauheim). Among 895 such cases, he found 121 with perceptible goitre. In 11 of these there were outspoken symptoms of Basedow's disease; in 110 there was goitre of long standing. The latter group was much the older, the ages ranging to 60 years; the small group of 11 all were young. Several authors, among whom are Bigler and Bauer, record the common occurrence of cardiac symptoms in endemic goitre.

PATHOLOGY

Before leaving the subject of the damaged heart, it may be well to touch briefly on the pathological aspects of the situation. Three of the series came to autopsy, the first showing nothing of consequence, the other two showing highly significant findings. These two cases, 11918-12340 and 13332, were both autopsied by Goodpasture[81] and meticulously described in the *Journal of the American Medical Association.* Goodpasture found that there were patches of acute myocardial damage, shown microscopically by hyalinization of some fibers, by a peculiar fraying out of most others. The fibrils were thinner, more widely separated, and seemingly torn across, their torn ends flattening to one side of the fiber. They were irregular, usually normally dark-staining at the periphery of the fiber and thin and light-staining at its center.

DIFFERENTIAL DIAGNOSIS

Hyperthyroidism is a general disease; hyperthyroidism is probably a toxemia of some sort dependent on thyroid abnormality; hyperthyroidism has marked cardiovascular symptoms. For these three reasons, it is often quite difficult to diagnose the disease from general disorders, from toxemia, and from cardiovascular diseases. Even in a full-blown case of hyperthyroidism, it may at times be difficult to say whether or not a valvular abnormality of the heart is present.

Neuro-circulatory asthenia (effort syndrome, soldier's heart) is probably the most difficult of all diseases to distinguish from an early light case of hyperthyroidism. Here are present all the subjective symptoms of exophthalmic goitre, such as palpitation, precordial pain, nervousness, excitement, weakness. Present, also, are various cardiovascular symptoms indistinguishable from those in hyperthyroidism,—tachycardia, lability of the pulse rate, vasomotor instability (easy blushing), a pounding heart with an accentuated first sound, possibly pulsating carotids, possibly a high pulse-pressure.

The relation, if any, between hyperthyroidism and neuro-circulatory asthenia is obscure. Possibly both are dependent on abnormality of the vegetative nervous system, hyperthyroidism being an advanced stage with the thyroid coming actively into play. Possibly, as some maintain, effort syndrome is hyperthyroidism in a mild form. Possibly the former is merely a neurasthenia.

At any rate, the diagnosis rests mainly upon objective symptoms such as exophthalmos, tremor, persistent tachycardia, cardiac irregularities and real cardiac damage; and mainly upon the pathognomonic sign of hyperthyroidism—an increased metabolic rate.

Mitral stenosis, as other authors such as White and Hamilton have pointed out, is difficult at times to distinguish from early hyperthyroidism. The frequent presence of a thrill; the loud, pounding first sound, suggesting very often a presystolic murmur; the presence of auricular fibrillation—all these make the observer think of the possibility of mitral stenosis. It should be remembered that hyperthyroidism is distinguished by forceful heart action and that arrhythmias are not uncommon. As White emphasizes, look for the diastolic murmur of mitral stenosis, and for the positive signs of hyperthyroidism. A rheumatic history is also important. Finally, it is not inconceivable that the one may be present together with the other.

Aortic regurgitation may occasionally be considered. It is not difficult to conceive this when it is remembered that practically all the symptoms and signs of aortic regurgitation, except the diastolic murmur, may be present in a case of undoubted hyperthyroidism. The forcefully acting heart causes a high systolic pressure, a resultingly high pulse pressure, pulsations of the carotids and femorals, pistol shots over them, and a Corrigan pulse almost as good as in aortic regurgitation. Added to this, if the observer is unaware of the frequency of heart-failure in hyperthyroidism, he may ascribe the whole cardiac condition to aortic valvular disease, present simultaneously with exophthalmic goitre. This may be mortifying if the case should come to autopsy, or if a question of treatment is involved.

Finally,—and, I believe, most important in the matter of diagnosis,—are those cases of undoubted cardiac damage, with atypical or few symptoms of hyperthyroidism. The picking out of these cases from the general run of heart-failure cases is somewhat of an art, dependent upon keen observation, upon the knowledge and belief that such cases occur, and upon a metabolic determination. Dr. Levine at this hospital has been most successful in "spotting" this type of case.

Especially in a goitre region, if a woman over 40 presents herself with cardiac symptoms—referable to the myocardium—cause unknown—one should look carefully for evidences of goitre, especially of the adenomatous variety. One should look for possible exophthalmos, for tremor, for pigmentation, and for such suggestive points in the history as periods of nervousness with loss of weight, attacks of palpitation, precordial pain, etc. Finally, a metabolism determination should be done in all cases where the possibility of hyperthyroidism is even remotely considered. I am sure that many a surprise awaits those who visualize the possibilities in this direction.

INTERPRETATION OF THE FOREGOING

Whether correct or no, I think it best to consider, at least in the case of the cardiac symptoms, that hyperthyroidism is a toxemia of some sort, probably closely related to an abnormal secretion, qualitative or quantitative, of the thyroid gland. I do not believe that a mechanical theory, or a theory which supposes that all the symptoms in hyperthyroidism are due to increased metabolism, would explain the cardiac symptoms. These *must* be explained, for it surely is no coincidence when in a large number of cases of hyperthyroidism, almost 30 per cent. are found to have hearts damaged in some manner. The theory that the consistently heightened metabolism puts a constant increased load on the heart, causing it to dilate, hypertrophy, and then weaken, cannot explain paroxysmal auricular fibrillation in early cases of the disease, nor does it explain early heart-block.

why not

It seems to me that if one would but hang one's hat on the hook of toxemia, temporarily at least, one could explain rationally all the various cardiac symptoms and signs occurring in the course of the disease. I will attempt to do so in the succeeding few paragraphs.

So-called normal hearts in hyperthyroidism are distinguished by a persistent tachycardia. Palpitation and precordial pain are common. Tumultuous action of the heart is the rule. The vasomotor system is unstable. Can these symptoms not be explained by assuming an irritation of the heart and vascular system by thyroid toxin?

Myocardial damage is common. It differs in nature according as to whether it occurs in young or in old, in severe or in mild, long-continued cases. In young people, with most of the myocardium normal, the irritation of the toxin, although severe, produces temporary acute damage, manifested clinically by paroxysmal auricular fibrillation, flutter, and tachycardia, by delayed conduction time and paroxysmal complete heart block. The age incidence of the paroxysmal arrhythmia and block groups is much younger than that of the heart-failure group. Long-continued, severe toxic damage, in persons who have not been operated upon, damages so much of the myocardium that it cannot recuperate, and the paroxysmal irregularities and blocks become permanent ones, as has been pointed out.

Paroxysmal block is interesting, showing probably that the toxin has hit the conducting system, temporarily blocking it.

Long-continued, mild toxic damage, occurring as it does in many endemic goitres, in adenoma with hyperthyroidism, "formes frustes," etc., would produce after many years as much cardiac damage as the severe toxemia of a full-blown case of hyperthyroidism acting over a shorter space of time. Thus, in old people, with mild hyperthyroid symptoms, there are apt to be severe cardiac signs.

The autopsy findings in the two cases of this series, reported by Goodpasture, bear out the contention of acute myocardial damage. In both these, acute areas of myocardial necroses, without valvular lesions, were discovered in cases of hyperthyroidism with hyperplastic thyroids.

SIGNIFICANCE OF THE OBSERVATIONS

The importance of these observations and analyses lies not alone in their value merely as records, but mainly in their application to treatment.

Arguing purely theoretically from the observations made above, I would naturally conclude that half-way measures have no place in a disease in which the heart is damaged so often and so severely. Medical treatment, in the form of rest, drugs, etc., may do more harm than good, since by a policy of watchful waiting, one may be eluded into thinking he has conquered the disease, when in reality cardiac damage is going on unseen all the while. The source of the toxemia must be removed, and quickly, and since this seems to be the thyroid gland, its secreting surface should be diminished considerably, either by surgery or by x-ray. The latter also has its disadvantages, it seems to me, since by depending too much on its efficacy and by carrying treatment with it over too long a period, one may find the patient sooner or later with a damaged heart. Operation thereafter would not cure the cardiac damage, though it would remove the source of its injury. Surgery or x-ray, then, are the only courses of treatment open in hyperthyroidism; and too much dependence should not be placed on the x-ray.

Endemic, or colloid goitres, and adenomata, should be dealt with, I believe, much more strictly than heretofore, because they undoubtedly lead in many cases, perhaps in most, to cardiac damage, if not to outspoken hyperthyroidism. Boothby says that "a patient of 23 years of age having an adenoma has a definite fixed chance of developing thyrotoxicosis during her 37th year." In the early years of life, medical treatment by iodides is probably of great value, but an adenoma seen in middle age, or even from 20 years on, should probably be removed surgically. In an elderly person with symptoms of cardiac damage, and mild hyperthyroidism, it is probably best to remove the adenoma and thus relieve the heart of the strain of continued toxic action.

SUMMARY

1. One hundred and forty-one cases, representing all the cases of hyperthyroidism in the Medical Service of the Peter Bent Brigham Hospital to the year 1922, inclusive, were studied with special reference to cardiovascular symptoms.

2. Ninety-five, or 67.3 per cent., were considered to have essentially normal hearts.

3. The characteristics of the heart normal for hyperthyroidism were studied.

(*a*) The subjective symptoms of palpitation and precordial pain were considered. Precordial pain occurred in 13.5 per cent. of the cases; in 10.5 per cent. of the normal, and in 22.5 per cent. of the damaged heart cases.

(*b*) The objective symptoms—such as loud, forceful first sound, systolic murmur, and thrill—were discussed. A thrill, presystolic or systolic, was noted in 30.5 per cent. of the cases.

(*c*) The objective vascular symptoms, such as pulsations of the peripheral vessels—carotids, femorals, brachials; "pistol-shots" over them; Corrigan pulse; capillary pulse; vasomotor instability, etc.—were mentioned.

(*d*) The question of blood-pressure was discussed, and it was decided that in practically all cases there is a higher systolic pressure than normal, and a high pulse pressure. The average blood-pressure was 146 mm. systolic, 72.5 mm. diastolic. In 25.5 per cent. of the cases, there was a hypertension (over 150 mm.).

(*e*) The electrocardiographic observations were touched upon. High T waves are characteristic.

4. Forty cases, or 28.5 per cent., were considered to have damaged hearts (myocarditis due to known causes such as rheumatic fever, nephritis, etc., excluded). These were divided into three classes:

(*a*) The paroxysmal arrhythmia group—12 cases. Of these, 9 had auricular fibrillation; 1, auricular flutter; 1, auricular fibrillation and flutter; 1, auricular tachycardia; average age, 39.5 years.

(*b*) The block group—5 cases: 2, paroxysmal complete block; 2, delayed conduction time; 1, bundle branch block; average age, 36.4 years.

(*c*) Heart-failure group—23 cases, with symptoms of heart-failure only. Of these, 10 had auricular fibrillation (permanent). Two types were distinguished: (1) the young, full-blown cases with high metabolism; (2) the oldish, mild, atypical case, usually with adenoma, and with relatively low metabolism.

5. Six cases, or 4.2 per cent., had hearts damaged by causes other than hyperthyroidism. Of these, 3 were mitral stenosis; 2 were syphilitic aortic regurgitation; 1 was a case of severe hypertension, with a heightened metabolism discovered accidentally.

6. The pathology of the heart was discussed shortly.

7. Diagnostic difficulties, and the differential diagnosis from neuro-circulatory asthenia, mitral stenosis, aortic regurgitation, and chronic myocarditis without known cause occurring in oldish people—these were discussed at length.

8. Lastly, suggested interpretation and the seeming significance of the various observations were set forth.

CONCLUSIONS

1. Forty cases with cardiac damage in 141 of hyperthyroidism represent no mere coincidence. This observation immediately puts the cardiovascular symptoms (complications, if you will) of hyperthyroidism above all other symptoms in importance.

2. All cardiovascular symptoms may well be explained by assuming that hyperthyroidism represents a toxemia of some sort, with especial predilection for the myocardium.

3. The heart which is not clinically damaged is probably one undergoing irritation from the toxin. This causes the subjective symptoms of palpitation and precordial pain, and the objective symptoms of rapid and forceful action.

The presence of forceful apex impulse, thrill, loud first sound, questionable presystolic murmur suggest mitral stenosis; the presence of peripheral pulsations and "pistol-shots," of high systolic pressure, Corrigan and capillary pulse suggest aortic regurgitation.

4. Damaged hearts probably represent myocardial destruction of more or less degree.

(*a*) Cases with paroxysmal arrhythmias, and with paroxysmal heart-block, occurring as they do usually in younger people, show a damaged heart muscle, with, however, enough good muscle for compensation.

(*b*) Cases with heart-failure, coming in persons below 40, are usually end-results of a full-blown hyperthyroidism with very marked toxic action over a short period.

(*c*) Cases with heart-failure in persons over 40 (usually over 50) are usually end-results of a mild or atypical hyperthyroidism, usually with adenoma, the toxic action of which has acted in small doses over a period of 20 years or more.

5. Suspect hyperthyroidism in cases showing paroxysmal arrhythmia; in young people with severe precordial pain or with attacks of palpitation; in women over 40 with heart-failure and thyroid adenoma.

6. Medical treatment, except preliminary to operation, has, I feel, no place in hyperthyroidism. Surgery (with x-ray as a poor second), by removing the source of the toxemia, is the only means for combating the disease. More attention, especially surgical attention, should be focused on adenoma of the thyroid. In middle-aged persons surely, in persons over 20 probably, it should be removed surgically.

7. I wish again to emphasize the paramount importance of constant watch over the heart in hyperthyroidism.

I wish to thank Dr. Henry A. Christian for his many courtesies.

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THE HYGIENE OF THE NERVOUS SYSTEM*

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THE problem about to be considered is a very broad one. It involves such subjects as economics, sociology and public morals quite as much as it does physiology and psychology.

Primarily, the hygiene of the nervous system is a matter of physiology. We are all possessed of a physical as well as a mental *ego;* indeed, unless we start with the conception that the physical conditions the mental, we place ourselves in the absurd position of assuming that the nervous system is exempt from the physiologic laws which govern the rest of the organism.

To understand thoroughly the important rôle played by the physical *ego* in the constitution of personality, we must first grasp the fundamental fact that Mind and Intelligence are by no means synonymous terms.† Mind is not wholly, or even mainly, intelligence. It consists largely, and in one sense entirely, of feelings. Feelings are in all cases the materials out of which, in the superior tracts of consciousness, Intellect is evolved by structural combination. Feeling is the substance of which, where it is present, Intellect is the form. Now, Feeling is itself decomposable into Sensation, although the character of a given feeling and that of the sensation which gives rise to it may be as unlike in kind and degree as can be imagined. By way of illustration, think of the feelings occasioned by the unexpected imprint of a cold hand on the back of one's neck in the dark. The unlikeness in kind and degree between the causative factor and its results shows us at once that Feeling is not to be judged by the sensational stimulus or stimuli which occasion it. It has a *flavor* all its own. If we lose sight of this fundamental fact we cripple enormously our power to achieve practical hygienic results.

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*The first of three lectures delivered in the spring of 1923 before the Department of Hygiene and Physical Education, Wellesley College.

†*Vid.* Spencer's Principles of Psychology; chapter on the composition of mind.

The nature of the physical *ego* is predetermined, and the factors which bring about this predetermination are not to be sought solely in our immediate forebears but also in a long ancestry, each generation of which has in some fashion wrought a modification in its nervous elements.

The *ego,* physically considered, is the entire organism and, as I have just said, the nature of this *ego* is predetermined. Further, this *ego* includes all the functions carried on by the structures of the organism when supplied with the requisite materials. These functions have for their net result the liberation from food and so forth of certain latent forces; and that distribution of these forces shown by the activities of the organism is from moment to moment caused partly by existing arrangements of its parts and partly by environing conditions. The physical structures thus pervaded by the forces thus obtained, constitute that substantial *ego* which lies behind and determines those ever-changing states of consciousness we call Mind. Given the external stimuli, and the nervous changes with their correlative mental states obviously depend partly on the nervous structures and partly on the amount of this diffused energy; each of which factors is determined by causes not in consciousness but beneath consciousness.

Biologic science teaches that both the development and stability of personality are primarily dependent upon the physical quality and the functional integrity of two great systems of nerves: (1) that which subserves what we term the "organic life"; and, (2), that which makes possible the "life of relation." Let us dwell for a time upon these two systems. The mechanism which underlies organic life is known as the autonomic nervous system and is composed of the sympathetic, the para-sympathetics and certain incretory glands termed "endocrine." Up to the present moment a satisfactory knowledge either of the exact physical structure or of the function of this complex neuro-glandular system is not at hand. If it were, the knowledge of the essence of life would be at our command, and biologic study would be at an end.

Of two facts concerning this most important neuro-glandular mechanism we are certain: (1) It nourishes both the nervous system and the vegetative organs, supplies them with the materials with which to carry on their functions and removes their waste products; (2) it represents the physical basis of the emotions. Without its aid the so-called life of relation would be seriously narrowed in its range and the upbuilding of personality materially stunted. In what way? This: the growth of Mind and Intelligence is directly conditioned by the diversity and multiplicity of our relations with the world about us. These relations are made possible through the harmonious coöperation of the motor or efferent and the sensory and sensorial or afferent elements of our nervous system. And these elements depend for their life and power upon materials brought through the blood stream by the neuro-glandular system above described. Our first notions of distance and weight spring from the feelings which come from muscular exertion; of solidity from the tactual and visual sensations derived from the multiplicity of avenues of approach to an object, made possible by concerted motor activities. In a word, our motor apparatus adjusts the photographic plates of our senses to outside impressions. These outside stimuli produce in us surface stimuli of the organs of common and special sensation. In the higher tracts of consciousness these sense stimuli become feelings which, as I have already shown, differ enormously in character from the occasioning stimuli. Those feelings constitute the mind, and those operations of the mind which have to do with a recognition of likeness or unlikeness in the perceived feelings are what we term intellectual. They constitute the mental *ego.* After a certain number of repetitions of the perception of a given feeling, its recognition is no longer an intellectual act but takes place in the lower levels of consciousness and becomes what is known scientifically as *intuitive.* This is all the more easily appreciated if we bear in mind that impressions received by the sensory components of mind are neither unsubstantial nor fugitive; but are, on the contrary, indelible.

In view of the physiologic foundation of personality, the importance of the rôle played by heredity in the determination of nervous stability cannot be over-estimated. Very early in the life of the person who has inherited a nervous system which is structurally and functionally of a high degree of vigor, there is established in the background of consciousness an immutable normal standard of *self-feeling.* It represents a fusion and sublimation of all the sensations streaming into the perceptive levels from the various parts of the organism. This *self-feeling* is, it is needless to say, extremely complex, and only measurably decomposable. It springs in part from the unrestricted excursion of the lungs in the respiratory act; from the combined action of vigorous heart muscle, intact cardiac valves and elastic arteries; from sound teeth; unhampered digestion; free elimination of waste products of every sort; a consciousness of potential vigor of the voluntary muscular system in its totality and a multitude of other sensations quite beyond the power of any available instrument of precision to detect, much less measure. Even the abundance and color of one's hair, the texture of one's skin and the freshness of one's complexion contribute certain elements to this complex *self-feeling* which is in reality an emotion. It is the joy of living, and it connotes freedom of action, power of self-expression, *amour propre*, self-reliance, and creative ability,—in a few words, all those egoistic feelings which are the basic elements in our awareness of being alive, and which govern our attitude toward our environment.

We are not from moment to moment entirely conscious of the existence of this vitally important and immutably standard norm of self-feeling, but that it is ever present in the background of consciousness is evidenced by our daily responses to enquiries concerning our health. The mere fact that we say: "I feel quite well," or "I do not feel quite up to the mark," betrays a conscious comparison on our part between self-feelings at the moment present in consciousness and our particular standard norm.

I have already called attention to the fact that impressions made upon the sensory elements of mind are permanent. This fact has a bearing upon heredity the importance of which cannot be over-estimated. If it is incontestable, —and biologic science assures us that it is—it follows that, where for a long time prior to procreation these sensory elements have been called upon to function inordinately, the imprint of this inordinate functioning becomes quite as indelible as that of the normal, and the product of the conception runs an enormous risk of inheriting a nervous system which is, to say the least, dynamically defective.

It matters little whether the causes of the parental abnormal nervous functioning are of internal or external origin. The mere struggle for existence may provide them. They may spring from poisons more or less subtle, voluntarily absorbed for the purpose of galvanizing jaded sense organs, or they may be furnished by toxins occasioned by germ infections. Whatever the cause, the chance of transmission of a nervous system over-prone to fatigue and to a morbid emotionalism tending to foster and increase this fatigue is very great.

Let us now examine into some of the ways in which individuals vitiate their nervous organizations and create a transmissible menace.

Sexual immorality causes annually an enormous wreckage of nervous health. Promiscuity in the sexual relation is almost certain to result in venereal disease in one form or another, the most deadly of which is that caused by the virus of the syphilitic spirochete. This virus is endowed with a special predilection for nervous tissues. Its devastating effect upon the rest of the organism are, to be sure, sufficiently ghastly, but that is a chapter by itself. What we need to know, in order to spread scientifically the gospel of nervous hygiene in the form of practical morality, is that the syphilitic poison enters the blood and is thus brought into the most intimate possible relation with brain, spinal cord and peripheral nerves.

The next great menaces to nervous, mental, moral and emotional stability are likewise circulating toxins—alcohol and the more villainous home-brewed products which, in spite of so-called national prohibition, are daily consumed in large quantities all over the country by representatives of every stratum of society from the highest to the lowest. Alcohol is a quickly diffusible stimulant. In excessive doses, even in its purest form, it causes chemical changes in nerve cells and fibers. In the crude and unspeakably impure form in which it is today literally poured into the human stomach, its toxic action upon the structures in question is enormously enhanced. All of us have many times witnessed the outward manifestations of an excessive ingestion of this poison, but have we thought of them in terms of nerve pathology? Up to a given point its action upon the brain tissues and nerves is a stimulating one. As it comes in contact with the frontal lobes it excites thought processes with such rapidity that one train is hardly started before another displaces it, with the result that confusion of thought results. Stimulation of the motor nerve apparatus betrays itself in volubility, gesticulation and leapings or dancing. When a certain degree of intoxication and over-stimulation is reached, temporary paralysis and incoördination ensue. The vasomotor or autonomic system betrays this stage by pallor, lack-luster eyes, vomiting and loss of sphincter control of the bladder. Ocular muscle palsy is evidenced by double vision. Through the sensory system equilibrium is destroyed and the gait is incoordinate. At this point the brain tissues are so drenched that events no longer register. Long before this stage is reached, however, moral inhibition is swept away and criminal impulses are immediately transformed into criminal acts. Alcohol is the open door to seduction, to promiscuity and to the dissemination of venereal disease.

When alcoholic poisoning is many times repeated, organic changes result. The peripheral nerves become inflamed. From this result extreme, persistent pain and tenderness of the nerve trunks, with associated muscular palsy and wasting. The effects upon the brain tissues are evidenced by loss of orientation as to time and place, hallucinations of memory and peculiar delusional states. In extreme cases a permanent dementia is observed. In the more moderate forms of chronic poisoning, a general lack of nervous tone is manifested by tremors and by a deterioration of the individual's moral fiber.

Another form of voluntary nerve poisoning deserves the most serious attention of the hygienist. One of the curses of the nervous habit is an over-active brain, and one of the most distressing results of this over-activity is insomnia. In practically every other household in the average community some member is today taking veronal, sulfonal or trional, or some combination of these potentially pernicious synthetic products, in order to induce sleep. The menace to nervous health of this widespread habit is very grave; nevertheless the law has not yet seen fit to restrict in any way the sale of these products. Let us understand their menace. To induce sleep they must, through the blood stream into which they find their way from the stomach, produce an anesthetic effect upon brain cells. This they achieve by temporarily altering the chemical composition of the blood which nourishes these cells. In last analysis, their effect upon these cells is akin to that of alcohol, and the observable manifestations of this effect are practically identical with those of chronic alcoholic poisoning.

Other poisons, such as opium, morphine and cocaine, if their use becomes habitual, produce mental disorders very marked in degree and most difficult of cure. Their effect upon the moral fiber is disastrous in the extreme. To satisfy a craving for these deadly nerve poisons, addicts will lie, steal and unblushingly prostitute themselves.

To a lesser degree the habitually excessive use of tea, coffee and tobacco leaves a detrimental imprint upon nerve tissues. The deleterious elements in tea are the alkaloid, theine, and tannic acid. Theine acts primarily as a stimulant to the autonomic system. After a time an organic longing for this stimulation is established. To satisfy it the tea drunk needs to be stronger and the amount consumed to be increased. If these demands are not satisfied, a peculiar tremor of the tongue and hands ensues and the heart-beat becomes arrhythmic. The tannic acid ultimately interferes seriously with the digestive processes and general nutrition suffers. Caffeine, the alkaloid of coffee, stimulates brain activity. We are all aware of its power to create undue wakefulness when coffee is drunk at night.

The poisonous effects of smoking tobacco are not, as is commonly supposed, due entirely or even mainly to the contained nicotine, but to the pyridine or combustion products which are gases. These gases inhaled into the lungs are quickly diffused through the blood stream and irritate the vasomotor apparatus. They may produce vertigo, coldness, mottling and sweating of the limbs and a serious interference with the digestive processes.

I speak at length of these voluntarily used poisons because their treacherous stimulating effects hold a tremendous lure for nervously adynamic people.

Now let us examine at length into the emotional trends and physical characteristics of individuals whose ancestral nervous systems have been vitiated in some one of the various ways I have endeavored to make plain to you. It is a common habit to speak of certain human aptitudes—let us say for art or music—as instinctive. In terms of physiology this means that in the nervous elements associative pathways subtending various orders of feelings have been rendered uncommonly permeable through a more or less long ancestry, hence the need of personal experience for achievements of various orders is reduced to a very marked degree. Now, in congenitally adynamic individuals this permeability of associative pathways is, as a rule, highly developed in a pathologic way; hence, the reactions of these individuals to stimuli, either from within or without, is excessive. Notably patent are the pathways which lead to the expression of the emotions, particularly to the most devastating of them all—fear.

It is quite beyond the power of words to give adequate expression to the potentiality of fear to annihilate the joy of living and to cripple useful activity. In the individuals we are considering, the most varied manifestations of its pernicious reactions are often discernible, even before the victims reach the stage of intellectual development which makes possible that synthesis of all the experiences and powers, past and present, which constitutes the conception of *self.*

Physically, these individuals show no particular abnormalities at birth, but they are easily thrown into convulsions by a breast-milk whose composition has been altered by maternal indiscretions in diet. And the same result may follow the eruption of teeth or the onset of one of the exanthematous disorders—scarlet fever or measles, for example. In these phenomena we have unmistakable evidence of the inherent weakness and the consequent irritability of the autonomic elements of the nervous system. Muscular and bony development seems to go on at a normal enough rate but the dimensions of the chest leave, as a rule, much to be desired. When the play-age arrives, we find no particular evidence of the too easy fatigability of muscles, which later becomes manifest unless the ministrations of the hygienist are invoked. Children of this type are generally fond of play, but enter into it with such abandon that bedtime finds them with dancing eyes, widely dilated pupils, burning cheeks and a brain so thoroughly overstimulated in every way that it is hours before sleep comes. If one watches them attentively in this sleep, it is very evident from the observed twistings and turnings and grinding of teeth, the intermittent jerkings of certain groups of muscles and the occasional outcries, that the night's so-called rest does not promise to repair the day's waste.

Such children are shy, diffident and, as has already been said, excessively timorous. If they are roughly handled in the course of play, they are much more apt to burst into tears than to retaliate in kind. Their imaginative powers—the term is here used in the popular sense—are entirely disproportionate to their years and to their experience with life. This abnormal play of fancy leads to frequent night-terrors.

Wherever one encounters a child who is abnormally shy, who bites his nails incessantly and exhibits regularly recurring contractions of certain groups of muscles, notably those of the face, neck and shoulders, it is safe to conclude that such a child has inherited an adynamic nervous system and needs sadly to be taken in hand by a person versed in the hygiene of that system. The muscular contractions in question are known as *tics*. They seem to be purposive—blinking of the eyes, grimacing, shrugging of the shoulders, for example—but they are not wholly so. Be this as it may, they invariably indicate nervous instability.

In school these children are apt to be quick in the intake, but appear at a tremendous disadvantage because of their incoercible diffidence and shyness. This is not generally apparent in the lower grades, but in the higher their plight becomes very evident. In the latter grades they are exposed to the ready mockery of more mature—hence, more cruelly critical—fellow pupils. The result is that, rather than stand up and recite before the class, they will answer, "Not prepared," even though they have the subject-matter of the lesson at their tongue's end. Directly they are forced to respond to the call to recite, their hearts beat to suffocation, they perspire excessively, their limbs tremble and their voices break. If they are taunted for these signs of weakness by their fellows, they are very apt to become tearful, and if this distressing situation recurs very many times, we find them beginning to withdraw into themselves, thus giving the first evidence of that anti-social tendency which, if not early checked, ultimately prevents their taking their proper place in the world.

With the advent of puberty there comes a crisis in the life of every boy and girl, whether the nervous inheritance be good or bad. The pernicious effects which so commonly follow this critical time are traceable to various causes which, in my opinion, are entirely preventable. The youth, finding himself in possession of procreative powers, of the real meaning of which he is but crudely aware, is very apt to experience certain lively emotional reactions which spring from this new possession. If these reactions are not intelligently explained to him, their pernicious consequences are, with deplorable frequency, very far-reaching and enduring. In ninety-nine out of every hundred cases of so-called functional nervous disorder—whether we term it neurasthenia, psychasthenia or hysteria—the bulk of the moral torture suffered is traceable to the crass ignorance of the individual with regard to the phenomena of puberty. Nature itself is an extremely bad counsellor at that disturbing time. It exercises no restraint whatsoever; on the contrary, it rather furthers the outcropping of sexual desires and in nowise inhibits their satisfaction. The frequent result is the establishment of the habit of masturbation, and in the latter's train there commonly follow terror and remorse—the most nerve-wasting of all the emotions.

In the maiden, the advent of menstrual life generally gives rise to a very complex state of affairs. Physically speaking it involves a new adjustment of the circulatory conditions of the organism, anatomical changes in the uterus and a new activity of the endocrinal or interstitial tissue of the ovaries. All these physical readjustments have an immense bearing on the self-feeling of which I have already spoken. Consciousness of sex is more fully awakened and, if excessive discomfort accompanies successive menstrual epochs, it is painfully awakened—a dread is established. But there are other and more subtle consequences. Curiosity is aroused, the inherent feeling of potential motherhood begins to take form, and sentiment and sentimentality are alike quickened. In certain instances Nature early proves imperious and gets the upper hand of innate delicacy, in which case the unhygienic habit already spoken of is established. This in turn leads to introspection and to the nervously devastating emotions also mentioned; in consequence, a vicious cycle is formed.

In the present post-war state of unrest, the social conditions have become so hectic that practically every hygienic law respecting the nervous system is set at defiance. The matter of character formation is permitted to settle itself. Before the heart-muscle has got its physiologic balance, adolescents are permitted to enter the most strenuous contests of physical endurance. Too great stress cannot be laid upon the folly of this.

Even in the higher social strata the dancing mania is productive of evil consequences, moral as well as physical. About the time sensible people are seeking the restorative virtues of sleep, the adolescents of both sexes of the above classes are beginning to proceed from theatres to ball-rooms. For the most part the attire of the young women is improper both from a moral and a hygienic standpoint. Dancing is kept up until the small hours of the morning. In the so-called "rest intervals" cigarettes and alcoholic beverages are more or less openly consumed and the digestive organs further taxed with indigestible foods of various kinds. So much for the physical drain of these performances. On the emotional side, the situation is no more hygienic, to say the least. The visual and tactual senses are unduly titillated and left in a state of disequilibrium. And all this takes place not once in a season but many times in several seasons. Small wonder that so many of these young people become nervously exhausted long before their coming out time. The years immediately preceding this event do not afford, hygienically speaking, an ideal preparation for the serious affairs of mature life.

In the workaday world there reigns a similar indifference to the laws of nervous hygiene. Here, as in the upper classes, adolescents, whether they are constitutionally prone to nervous exhaustion or not, subject themselves to the same depleting influences; and it is not by any means in factories or workshops that these influences are encountered. The majority of the present-day industrial plants are constructed with a view to the admission of an abundant supply of fresh air and sunlight, and the desirability, from an economic standpoint, of maintaining the nervous vigor of employees is thoroughly appreciated by Capital. The labor unions see to it that the hours of toil are not unduly prolonged. No, the menace to nervous stability among the laboring classes is in part hereditary in origin, in part the result of the unhygienic character of the home conditions and in part the outcome of aping the more well-to-do in the pursuit of vice in the guise of pleasure.

Formerly the neurologist used to encounter many cases of poisoning by lead, arsenic and other mineral poisons in some way taken into the system during the course of industrial pursuits. The spread of the gospel of hygiene has diminished the number of these cases to a marked degree. The incessant over-strain of certain coördinated groups of muscles will produce paralysis and wasting of these muscles, and may even cause organic decay in certain cell-groups in the spinal cord, from which the nerves which energize and nourish such muscles spring. These results come under the general head of occupational neuroses. Through the spread of hygienic doctrines, they likewise are growing to be things of the past.

It remains, finally, to inquire as to how either the inherited or acquired adynamic state may eventuate unless early recognized and brought under the beneficent influence of hygiene. Let us think in terms of anatomy and physiology rather than in the language of psychology. Pathologic exhaustion of the autonomic system manifests itself in two ways at least: ~~(1) in~~ an ~~individual's emotional~~ life; (2) in his vegetative functions. And, inasmuch as the two are most intimately associated, functionally speaking, a vicious cycle of pathologic activity is early established. Any constituent element of the nervous system which becomes adynamic becomes at the same time irritable; hence, in the particular asthenic condition under discussion we must expect the most varied alternation of manifestations of over and under-functioning. Let us start with the vegetative functions of the autonomic system. As a result of incessant quantitative variations in the functional activity of this system, the mouth may be at one time as dry as parchment; at another the saliva may flow so freely that an individual actually drools. At one period the stomach is hypoacid and little or no pepsin is secreted; then the conditions may suddenly shift, hyperacidity replaces its opposite, and the burning sensation which ensues in the stomach and esophagus suggests that those tissues are digesting themselves. Both in the hypo- and hyper-acid states the sufferer is painfully aware of the digestive process from start to finish, and by no means the smallest element of his distress is the gas which distends the stomach and intestine, crowds the heart, whose rhythm it seriously disturbs, and adds enormously to the terror and apprehension from which the victim habitually suffers. Constipation and diarrhea alternate. Frequent micturition is a commonplace; the urine is apt to be either of watery paleness or of a brick-red color. Excess in its phosphatic content alternates with excess in its holding of urates. The body temperature is practically always subnormal, and the slightest rise of temperature causes discomfort. The heart at times beats to the point of suffocation; at times its pulsations are hardly perceptible at the wrist. The genitals become flabby and cold. Menstruation may cease for protracted periods or it may exhibit marked irregularities as to amount and as to associated pain. Skin circulation shows precisely similar variations. At times the extremities are cold, mottled and sweaty, at times burning hot.

This is not the complete story of disturbed vegetative function, but it is enough to explain the alteration of the resultant self-feeling which inevitably ensues. By contrast with the standard normal self-feeling of the individual, the new is horrifying in the extreme, and a consciousness of a cleavage of personality gradually results. To my mind this explanation of the origin of a doubling or a multiplication of the personality is much more intelligible and logical than those based on purely psychologic data. Be this as it may, the depression which springs from the consciousness of the cleavage in question is profound, and leads to other emotional reactions of the most extraordinary character. A complete description of them is here impossible. They all react intensely upon the vegetative functions and make them even more feeble and irregular.

The motor elements of the nervous system betray their participation in the adynamic disorder by a constant feeling af fatigue and muscular impotency. Even after a night's rest, the victim awakes "all tired out," and begins the day with a sensation of having to lift the entire universe with every respiratory movement. It is a task to dress, to bathe, to do the hair. The jaws tire excessively during a meal, the voice grows faint in the course of even a short conversation, and the writing of a letter ends in a more or less unintelligible scrawl. A short walk necessitates a long rest.

On the sensory side all sorts of perversions are manifest—curious feelings of emptiness, fullness, weight or tightness in and about the head; tenderness of the spine, particularly emphasized in the cervical and coccygeal regions; numbness and prickling in the extremities, which "go to sleep" quickly when kept in contact with a hard surface for any length of time. The picture is one of so many details that I cannot possibly paint it in its entirety.

What I hope to have made clear is that the victim of the riot of function which goes on in the economy as the result of a pathologic exhaustion of the latter's constituent nervous elements furnishes the practical hygienist with a problem whose solution is by no means to be sought in mere verbal formulae. I hope also to have made clear, even though by indirection, that the work of salvage must be begun at an early period in a menaced individual's career. There are two aspects to this work—the one physical, the other psychologic. At the next conference I shall endeavor to outline the physical.

Medical Progress

PROGRESS IN THE STUDY OF CARDIOVASCULAR DISEASE DURING 1923

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I. PREVENTION

It is in accordance with the spirit of the times to begin a report on the progress of cardiovascular disease during 1923 with a discussion of what has been accomplished for the prevention of heart disease. Since the organization of the associations for the prevention and relief of heart disease in New York, Boston, Philadelphia and Chicago, a new interest has arisen in the etiology and epidemiology of rheumatic infections and in the economic problems of the "cardiac cripple." Studies on the family incidence of rheumatic infections similar to those made by St. Lawrence in New York (*Jour. Am. Med. Assn.*, 78:251, and *Jour. Am. Med. Assn.*, 79:2051) are being carried on in other cities of America. In October of 1923, at a meeting of the Section of Medicine of the British Medical Association, a symposium was held on the etiology and treatment of heart disease in early life, with especial reference to the prevention of chronic cardiac insufficiency (*Brit. Med. Jour.*, Oct. 20, 1923, p. 702). The opening paper was given by Reginald Miller, who discussed every phase of the subject in the light of present knowledge. He suggested that there may be a definite relation between the incidence of rheumatic heart disease and poverty. He also asks the questions: "What is the origin of the repeated relapses and reinfections in the rheumatic state and how can they be prevented?" and "How can we be enabled to give long courses of treatment to our many cardiac cripples in the hope of prolonging their lives?" Twelve men from various parts of the British Isles told of their experiences with rheumatic heart disease and the meeting recommended that "a special committee be appointed to consider the best steps to be taken to combat the grave menace to the community arising from chronic cardiac disease." Thus the forces of prevention of heart disease are being marshalled on both sides of the Atlantic.

The analysis of findings in 100 cases of so-called effort syndrome by McCrudden and White (BOSTON MEDICAL AND SURGICAL JOURNAL, 188:64) is of interest in relation to the prevention of heart disease: 45 per cent. were sent to the hospital with a diagnosis of valvular heart disease or myocarditis. None of these cases showed any evidence of organic heart disease. Proper regard for the criteria of organic heart disease is essential in diagnosis, especially in the presence of symptoms suggesting effort syndrome.

From an analysis of 470 cases of mitral stenosis Willius (*Ann. Clin. Med.*, 1:326) concludes that a great number of deaths in patients with this lesion occurs in the sixth and seventh decades. Betchov (*Arch. d. Mal. d. Coeur*, 16:646) analyzed 115 cases of inflammatory valvular affections, mostly rheumatic, and found that the average survival period after the beginning of valvular disease is 19.8 years. In diseases of mitral valve alone the average survival period is 23.9 years; of aortic valve alone, 19.2 years; and when both valves are involved, 15.1 years.

Pratt (Med. Clin. N. Am., 5:1305, March, 1922) has called attention to the infrequency of rheumatic fever and rheumatic heart disease in the southern part of the United States. This observation is of course significant in respect to the etiology and possibly the prevention of this type of heart disease.

The recurrence of cardiac infections in children with chronic valvular disease was studied in a series of 80 children followed for three years by Smith and Stulik (*Am. Jour. Dis. Child.*, 26:391). Twenty-one had frequent periods of slight fever ranging from 99.2° to 100.4° F. and in 16 of these a diagnosis of active cardiac infection was made. In seven instances the diagnosis was based upon a conduction defect in the auriculo-ventricular bundle. The rate of progression of the cardiac disease was much more rapid and the incidence of severely damaged hearts was far greater amongst these 21 children than in the remaining 59. These investigators recommend education of the public, the early elimination of oral and nasopharyngeal infections and the establishment of convalescent homes.

The question of the influence of tonsillectomy in acute rheumatism in children has had some new light thrown upon it by the investigations of G. H. Hunt (Guy's Hosp. Rep., 73:383, *Jour. Am. Med. Assn.*, 82:231). He sent letters to 250 patients who had had acute rheumatism asking for information as to the frequency and nature of recurrences. Of these only 144 supplied satisfactory data. The analysis of this group of cases reveals the following significant facts: Of the 66 in which enucleation was done, 35 had recurrences; no operation was performed in 78, and 33 of these had recurrences. Ninety-six had been admitted to the hospital with the first attack of acute rheumatism; recurrences were reported by 38 of these. The tonsils were enucleated in 50 of this group of 96 and recurrences occurred in 23. Of the remaining 46 in which no operation was done recurrences were reported by 15. Forty-eight were admitted for a second or later attack and recurrences occurred in 30; enucleation was performed in 16, and 12 of these patients had recurrences; of the other 32, recurrent attacks were reported by 18.

These data seem to indicate that recurrence is more likely to occur after a second than after a first attack. In both groups recurrence was somewhat more frequent in cases with tonsillectomy, but there is not any justification for the positive conclusion that the operation increases the liability to recurrence. The figures are, however, suggestive.

In 39 of the 74 cases with heart disease in this group of 144 patients, there were recurrences of rheumatic fever, while in the other 70 cases recurrence took place in 29. It seems, therefore, that the presence of heart lesions increases the frequency of recurrences.

This investigator concludes that whereas it is justifiable to remove the tonsils in cases with local signs of inflammation it is clear that in rheumatic children whose tonsils and lymphatic glands appear healthy, tonsillectomy is not indicated and is an unjustifiable operation.

These observations are of great significance. Since the beginning of the present great wave of frequent tonsillectomies, many surgeons who do the operation and physicians who advise it must have large groups of cases in which they could make similar studies to that of Hunt. At the present time, when the questions of prevention are uppermost in the minds of both the profession and the public, such studies are urgently necessary.

Bloomfield and Felty have continued their investigations of the etiology and epidemiology of nasopharyngeal infections. Bloomfield (Johns Hopkins Hosp. Bull., 34:65) studied the effects of antiseptics on the bacterial flora of the upper air passages and found that in spite of the fact that some of the drugs, notably mercurochrome, penetrated into the mucous membrane and the tissues beneath it, the bacterial flora remained practically unaffected. It is possible that whatever benefit is observed clinically from the use of antiseptics may be due to the promotion of drainage by shrinking of the mucous membranes.

Basing their rationale on evidence which showed that carriage of beta-hemolytic streptococcus in the tonsil is associated with a high degree of resistance to tonsillitis, Bloomfield and Felty (Johns Hopkins Hosp. Bull., 34:251) made attempts at vaccination for the prevention of tonsillitis. Both carriers and non-carriers were vaccinated and it was found that carriers were free from tonsillitis (whether vaccinated or not) and in non-carriers the protective power of the inoculation was suggested by the lower incidence of tonsillitis among the vaccinated and by the extremely mild form of the disease in those who did develop infection.

In a report on the dissemination of hemolytic streptococci among a group of healthy people the same investigators (Johns Hopkins Hosp. Bull., 34:414) draw attention to the necessity of differentiating between "hemolytic streptococci of the true disease-producing or beta type and the very similar but essentially nonpathogenic alpha prime type (Brown)."

The work of these investigators (*Arch. Int. Med.*, 32:483) on the bacteriology of acute tonsillitis with reference to epidemiology and susceptibility has led them to form certain significant conclusions. In the series of cases which they studied, acute follicular tonsillitis was uniformly due to the beta-hemolytic type of streptococcus. Following the disease, these organisms were found to persist in the tonsils for an indefinite length of time. From the analysis of the epidemiological evidence in this series of cases it appears that the outbreak of tonsillitis was not an epidemic but rather a group of sporadic cases. In their opinion tonsillectomy confers a high degree of protection against acute streptococcic infection of the lymphoid tissue of the throat. A certain number of persons in the group studied seemed naturally insusceptible or relatively insusceptible to tonsillitis. Tonsillitis as a rule is not an autogenous infection but seems to occur in individuals not already carriers of hemolytic streptococci.

II. SURGICAL TREATMENT OF HEART DISEASE

Jonnescu's report, in 1920 (*Bull. d. l'Acad. d. Med.*, 84:93), of a case in which he resected both lower cervical ganglia and the first thoracic on the right and left sides for the relief of angina pectoris with the result that the patient was free from attacks for four years after the operation in 1916, stirred up much interest in the surgical treatment of this condition. Coffey and Brown (*Arch. Int. Med.*, 31:200) report five cases of angina pectoris in which sympathectomy was performed only on the left side and consisted of removal of all three ganglia in one case and only the two cervical in the remaining four. Their results are encouraging in that four patients had practically no symptoms two to seven months after the operation. Jonnescu (*Bull. d. l'Acad. d. Med.*, 90:168) reports six cases in which he performed his original operation. Two died a few days after the operation from cardiac failure, one died eight months after, and three, including the 1916 case, are cured. Brünning (*Deutsch. med. Wchnschr.*, 49:945) also reports a case of "cure" of angina pectoris of the "vasomotor type," in which he performed Jonnescu's operation.

A significant advance in cardiac surgery is reported by Elliott C. Cutler and S. A. Levine (BOSTON MEDICAL AND SURGICAL JOURNAL, 188: 1023). Apparently for the first time a successful operation of valvulotomy was performed on a human subject. The patient, a girl of 12 with marked mitral stenosis, sustained the operation very well and will be followed with great interest. The character of the murmurs was changed by the operation so that the diastolic murmur became less, and the systolic more marked than before valvulotomy, thus indicating an increase in size of the mitral orifice.

III. QUINIDIN SULPHATE THERAPY

During the past few years the therapeutic value of quinidin sulphate has been quite thoroughly investigated and numerous writers have reached similar conclusions as to the indications and contraindications for the use of this drug. Viko, Marvin and White (*Arch. Int. Med.*, 31:345) report the largest series, 71 unselected cases of chronic fibrillation and four of nonparoxysmal (chronic) flutter. Sixty-eight per cent. of this series were restored to normal rhythm, and in 34 per cent. it was maintained for at least ten months. In a smaller series of 16 selected cases, Burwell and Dieuaide (*Arch. Int. Med.*, 31:518) report restoration of normal rhythm in 14 of 16 cases, with permanent result in eight and temporary result in 6. Both these groups of investigators employed about the same doses, namely "a test dose of 0.2 gram twice (with a two-hour interval) on the day preceding full dosage" . . . then "0.4 gram five times daily by mouth . . . at two-hour intervals, in some cases increasing to 0.6 or 0.8 gram five times daily. Usually the drug was continued until restoration of normal rhythm or development of significant toxic symptoms" (Viko, Marvin and White). Numerous other reports have been made by writers in America and in Europe. Examination of ten representative reports including 355 cases of chronic fibrillation and flutter treated with quinidin reveals the following points on which there is almost uniform agreement: (1) Patients of different ages respond about equally well to quinidin. Clark-Kennedy (*Quart. Jour. Med.*, 63:204) states that the middle-aged respond better than youths. (2) The presence or absence of a valvular lesion is not an important factor in the response to quinidin. Clark-Kennedy adds that in cases of exophthalmic goiter better results are obtained during non-toxic state following thyroidectomy or natural remission. (3) Cases with fibrillation of short duration (under six months) give the best results. (4) Before beginning the administration of quinidin it is preferable to reduce the apical rate to 80 by digitalization. (5) Cases of congestive failure are more apt to relapse. (6) Small hearts respond more readily than large hearts. (7) Embolism and sudden death (most likely also due to embolism) are apt to occur in cases with fibrillation of long duration and in those in which there is or was objective congestive failure. It is in these types also that normal rhythm is most apt to be only temporary when restored by the use of quinidin. Therefore in such cases the drug seems contraindicated.

Some investigators stopped the administration of quinidin as soon as normal rhythm was established, while others prescribed a maintenance dose of about 0.4 gram per day. The great majority of cases were treated while in the hospital.

Waldo B. Farnum (*N. Y. State Jour. Med.*, 23:15) reports results in a series of 20 ambulatory cases of fibrillation previously well digitalized; the doses he employed did not exceed 10 grains (0.6+ gram) per day. Only three cases were restored to normal rhythm, and of these one reverted to fibrillation within a few days while the other two maintained sinus rhythm for at least six weeks. The author considers these results unsatisfactory and concludes that quinidin is not effective in patients such as he describes. It is obvious that the doses he employed were relatively small, and it may be that better results could have been obtained with larger doses even in ambulatory patients.

Viko, Marvin and White, and Burwell and Dieuaide used larger doses than those generally employed by other workers. Furthermore, whereas these authors gave 2.0 grams on the first day after the test dose others gradually increased the dose, *e.g.*, beginning with 0.2 gram, they increased by 0.2 gram daily until 1.2 gram a day was administered. Most investigators have not exceeded 1.2 grams a day. In general it appears that better results were reported by those who used larger doses. Finally, it seems that the policy of the first groups of investigators, as quoted above, is the most rational.

Clinical observers agree that it is advantageous to the patient to have normal rhythm rather than fibrillation controlled by digitalis. Further evidence in favor of this view is presented by Stewart (*Arch. Int. Med.*, 31:871), who made blood gas observations enabling him to measure blood flow before and after restoration of the normal mechanism in cases of auricular fibrillation. During fibrillation oxygen capacity was increased due to increase in amount of hemoglobin content of the blood, and there was also some increased oxygen consumption: the oxygen saturation of the blood was however lowered. With restoration of normal rhythm the normal balance of these factors was also restored. The same worker, together with Edward P. Carter (*Arch. Int. Med.*, 31:900) reports similar results in a case of paroxysmal tachycardia. It appears therefore that during fibrillation and also during paroxysmal tachycardia there is diminished blood flow and the increase in oxygen capacity is an adaptive mechanism to compensate for the decreased oxygen saturation.

The evidence found in the literature of 1923 favors the view that in suitable cases of auricular fibrillation it is advisable to give quinidin sulphate in sufficient amount to restore normal rhythm.

Iliescu and Sebastiani (*Heart*, 10:223) have made some interesting observations on the action of quinidin in paroxysmal tachycardia. In a case of frequent short paroxysms they observed that as quinidin slows the paroxysmal rate the paroxysm becomes longer and continues until the normal rate of the same heart is reached, when normal rhythm becomes established. When the heart ascapes from the effect of the quinidin the events occur in the reverse order.

C. E. Wolferth (*Ann. Clin. Med.*, 12:123) describes a case with frequent paroxysms of tachycardia in which a gradual increase in the dose of quinidin was accompanied by corresponding diminution in frequency of attacks; it required 3.0 grams per day to cause complete cessation of the attacks. Then, on diminishing the dose, paroxysms appeared again. A maintenance dose of 0.4 gram four times a day was necessary to keep this patient free from attacks. Viko, Marvin and White report treatment of eight cases of paroxysmal auricular fibrillation and one of paroxysmal auricular flutter, and recommend the administration of 0.4 gram of quinidin sulphate per day.

Isolated cases of embolism are described in unselected series of cases with auricular fibrillation, but with careful selection it is reasonable to expect that this accident can be effectively avoided. Kerr and Bender (*Heart*, 9:26, 1922) describe attacks of syncope accompanied by probable ventricular fibrillation. The patient recovered and remained well. In 484 cases treated with quinidin analyzed by Viko, Marvin and White (*ibid.*) 15, or 3.1 per cent., died of proven or possible embolism and seven from other causes, including one case of fixed flutter.

IV. CHANGES IN SIZE OF THE HEART IN RESPONSE TO FUNCTIONAL DEMANDS

Friedman and Strauss (*Arch. Int. Med.*, 32:601) observed 21 cases of cardiac decompensation during the progress of compensation and, using x-ray methods, noted no change in 35 per cent., decrease in all diameters in 35 per cent., and decrease in right border only in 30 per cent.

Strong and Gordon (*Arch. Int. Med.*, 32:510, 517) studied changes in size of rabbits' hearts before and after exercise, using x-ray methods of measurment, and found that in both normal hearts and those in which degeneration was produced by injection of spartein and adrenalin chloride, the heart diminished in size immediately after exercise.

Gordon has recently reported at a meeting of the Harvard Medical Society in Boston (December 11, 1923) his results of an x-ray study of the hearts of Marathon runners immediately and again several hours and several days after the finish of the Ashland to Boston annual Marathon race (25 miles). He discovered that the hearts of these strenuous athletes were of normal size even in men who had run for years, and that they tended to be smallest of all immediately at the finish of the race. In the past erroneous conclusions had been sometimes made from the physical examination of such runners.

These contributions are mentioned here because of their value in throwing some light on the problem of changes in size of the heart with variations in function. More such exact data are needed in order to clarify this subject.

Robert L. Levy (*Arch. Int. Med.*, 32:359) made teleroentgenographic studies in 21 cases of pneumonia and eight cases of bronchopneumonia, and found that cardiac dilatation occurred in 61.9 per cent. of the cases of lobar pneumonia and in 25 per cent. of those with bronchopneumonia. Dilatation was less frequently observed in cases of lobar pneumonia who received digitalis (50 per cent.) than in those who did not (72.7 per cent.). Those with bronchopneumonia who were given digitalis showed no dilatation. Arguing that dilatation is evidence of the attempt of heart muscle to augment systolic output, on the basis of Starling's theory that there is increased force with lengthening of the muscle fibers, Levy concludes that patients with pneumonia should be treated with therapeutic doses of digitalis in the presence of normal rhythm as well as during auricular fibrillation or flutter. A similar study of a larger series of cases would be essential in establishing the significance of these observations more conclusively.

V. ENDOCARDITIS

In an article on the "characterization of various forms of endocarditis," Libman (*Jour. Am. Med. Assn.*, 80:813) differentiates between the rheumatic, syphilitic, acute bacterial, subacute bacterial and indeterminate groups of endocarditis. The last consists of two kinds, the so-called terminal type and an atypical verrucous form of endocarditis which is quite a definite clinico-pathological entity, but for which no definite etiology is known. Dr. Libman also throws new light upon the life history of subacute bacterial endocarditis. He describes five cases of recovery from this disease which was hitherto considered invariably fatal. Furthermore, he describes recurrences of the disease in cases which passed through the first attack without noticeable symptoms and signs. He suggests that mild forms of subacute bacterial endocarditis may be much more common than is at present realized. In concluding, Libman states that "it will be of greatest value if an active campaign is undertaken for the purpose of preventing this as well as other forms of endocarditis."

From an extensive, thorough investigation of the incidence of congenital bicuspid aortic valve in relation to the occurrence of subacute bacterial endocarditis, Lewis and Grant (*Heart*, 10:21) conclude that this valvular abnormality is a determining factor in trapping bacteria and thus favors the development of subacute bacterial endocarditis.

Blumer's monograph (*Medicine*, 2:105) on subacute bacterial endocarditis is a valuable contribution to the literature on this subject. A number of reports of series of cases of subacute bacterial endocarditis are to be found in the literature for 1923; evidently this disease is being more commonly detected.

VI. CORONARY THROMBOSIS

The clinical syndrome associated with this anatomical condition is not one that is very distinct and clear-cut; the symptoms and signs are almost as variable as those of subacute bacterial endocarditis. Therefore, the careful analysis of the symptoms, signs and necropsy findings of 19 cases of coronary thrombosis by Wearn (*Am. Jour. Med. Sc.*, 165:250) is a valuable contribution to the study of this condition. From this study Wearn reconstructs a summary of the clinical features of this condition in which he draws attention to the fact that usually the attack of severe pain over the heart, lower end of the sternum or in the epigastrium associated with marked prostration may be the first sign of heart trouble in the patient. Sometimes dyspnea rather than pain is the presenting symptom. The attack of pain or dyspnea or both is not a spasmodic one, such as "angina pectoris," but persists, and sedatives in the usual doses often fail to bring relief. The general appearance of the patient is that of a state of severe illness, a weakly acting heart, normal rhythm or irregularities due to premature beats, often heart block and occasionally a pericardial friction rub. Tenderness in the region of pain, an enlarged tender liver and some fever with occasionally a polymorphonuclear leucocytosis are the principal signs that are to be looked for. The disease is not necessarily fatal.

Two typical cases of coronary thrombosis occurring in two brothers are described by David Reisman (Med. Clin. N. Am., 6:861). In each of these there was no pulsation in the dorsalis pedis artery, and the mental state was one of an "overwhelming fear of death or the settled conviction of dissolution." This writer points out that the attacks may resemble those of "ptomaine poisoning," and in obtaining a history of previous attacks this description is of value.

From a study of eleven cases of coronary thrombosis and from the reports of experimental investigations on coronary occlusion Fred M. Smith (*Arch. Int. Med.*, 32:492) concludes that in a large percentage of such cases significant changes in the electrocardiogram occur in the first few days following the onset of the cardiac accident. In some instances these changes may persist for weeks. Alterations in the T deflection and an alteration of the Q.R.S. group are the most constant findings. More of such reports as these will contribute greatly to the wider knowledge of this condition and its more frequent detection.

VII. HEART PAIN

The exact nature of the cause of heart pain still remains a debatable subject. However, much has been added to the study of this symptom. A contribution has been made by Paul D. White and J. Edwin Wood, Jr., in their classification of heart pain which is summarized as follows: (1) simple fatigue pain which occurs in (*a*) chronic hypertension, (*b*) aortic stenosis or regurgitation, (*c*) mitral stenosis, (*d*) pulmonic stenosis—congenital heart disease, (*e*) adherent pericarditis, (*f*) paroxysmal tachycardia or paroxysmal auricular fibrillation or flutter, (*g*) permanent auricular fibrillation or flutter with high ventricular rate, (*h*) permanent coronary narrowing due to arteriosclerosis; (2) nervous heart pain, including effort syndrome; (3) paroxysmal heart pain (probably of coronary disease or irritability), the so-called true angina pectoris: (4) pain of coronary thrombosis; (5) aortic pain, of syphilitic aortitis and aneurysm; (6) pain of pericarditis.

It is noted also that frequently combinations of these factors occur in producing heart pain. The adoption of such a classification should lead to a clearer understanding of the symptoms complained of by patients and also make discussions on heart pain more intelligible.

The school of Sir James Mackenzie, working at St. Andrews, has published reports of clinical investigations of the nature of symptoms, heart pain included.[*] In his book on "Angina Pectoris" (Oxford Med. Pub., 1923), which appeared recently, Mackenzie deals in detail with the principles underlying these investigations. He applies the principles of the reflex arc in the explanation of the pain of angina pectoris. He still maintains his view that this symptom is but a symptom and that it may be caused either by organic changes in the heart or by an undue susceptibility of the nervous system. He also states, as in previous writings, that deficient blood supply to heart muscle due to coronary disease is the chief cause of angina pectoris due to organic heart disease. A valuable part of this book is the appendix containing the reports of 160 cases of angina pectoris including the records of 22 autopsies.

Gallavardin (*Paris Med.*, 13: 16) reports 15 cases, in addition to the 10 he reported two years ago, of "angina pectoris" occurring in mitral and aortic stenosis. The interpretation of such severe pain in rheumatic heart disease is of much interest. In our experience it has resulted from the fatigue of paroxysmal tachycardia so common in these cases or from the fatigue found with early congestive failure, or from effort syndrome.

VIII. CONGENITAL HEART DISEASE

The application of blood gas studies to the investigation of the abnormal physiology in cases of congenital heart disease has brought to light some very interesting and significant facts. In a monograph on "Cyanosis" (*Medicine,* 2:1), Lundsgaard and Van Slyke present an analysis of the various factors which contribute to the causation of cyanosis, namely, diminished oxygen saturation due to impaired pulmonary aeration, slowing of the peripheral stream, increase in total hemoglobin and shunting of the venous blood into the arterial stream through some mechanical defect in heart or great vessels or by the passage of blood through lung tissue which is completely deprived of its air supply. This last factor of right to left shunting of the blood is the chief cause of cyanosis in cases of congenital pulmonic stenosis when complicated by interventricular septal defect and *rechtslage* of the aorta. In such cases there is also stasis in the peripheral capillary stream and thus these two factors are the cause of cyanosis in the principal type of "morbus coeruleus." In other defects such as simple defect of the interventricular septum or patent ductus arteriosus, because of higher pressure in the left than in the right side of the heart a left to right shunting of blood prevails: this merely somewhat overloads pulmonary circulation but does not cause cyanosis. If, however, as Maude E. Abbott has pointed out in the section on Treatment of Congenital Heart Disease which is to appear in the new Forscheimer's System of Therapeutics and in a lecture on this subject before the Boston Association for the Prevention and Relief of Heart Disease on January 17, 1924, for some reason such as pneumonia or emphysema the pressure in the right side of the heart is sufficiently raised to cause a right to left shunt (of at least one-third[*]) of the pulmonary circulation, then cyanosis will occur. In such cases also the other factors of peripheral stasis and high hemoglobin content may contribute to accentuation of the cyanosis. These observations form an important contribution to the pathological physiology in cases of congenital heart disease.

The literature of 1923 contains an unusual number of interesting clinical and morphological studies in congenital heart disease. Abbott, Lewis and Beattie (*Am. Jour. Med. Sc.*, 165: 636) contribute a differential study of a case of pulmonary stenosis of inflammatory origin (ventricular septum closed) and two cases of (*a*) pulmonary stenosis and (*b*) pulmonary atresia of developmental origin with associated ventricular septal defect and death from paradoxical embolism. The ages in these three cases were 14, 10 and 11 years respectively. The clinical features are carefully analyzed and correlated with the morphological findings.

Clarke (*Jour. Am. Med. Assn.*, 81:371) reports a case of bacterial endocarditis of the aortic cusp over a large defect in the interventricular septum. The significant observations made by Sir Thomas Lewis and Ronald T. Grant on the relation between congenital bicuspid aortic valve and the incidence of endocarditis have been discussed above in the section on subacute bacterial endocarditis.

Bishop (*Jour. Am. Med. Assn.*, 80:547) draws attention to the presence of high systemic blood pressure in certain cases of congenital heart disease without cyanosis. This hypertension is explained as a means of compensating for the high pulmonary pressure, thus maintaining a left to right shunt of the blood stream.

P. Meyer (*Arch. d. Mal. d. Coeur,* 16:16) describes a case of transposition of the heart and viscera with congenital constriction of the pulmonary artery, interventricular septal defect and complete heart block in a child which lived for four and one-half months. Fifteen other cases of congenital heart block are referred to.

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[*] P. T. Herring: Brit. Med. Jour., Part 1, Oct. 6; Part 2, Oct. 13; Part 3, Oct. 20; Part 4, Oct. 27, 1923. "The Regulating and Reflex Process."
Sir James Mackenzie: Lancet, Vol. ccv, Part 1, Nov. 3; Part 2, Nov. 10; Part 3, Nov. 17, 1923. "The Principles of Symptomatology: An Introduction to a New Outlook in Medicine."

[*] Lundsgaard and Van Slyke: "Cyanosis," Medicine, 2:1.

This observer believes in the view that the heart block is not due to congenital malformation but is the result of inflammatory changes in the region of the septal defect, such congenital malformations being predisposing factors to infection. Other authors, however, notably Maude E. Abbott (personal communication), believe that complete heart block may occur independent of inflammatory changes in cases of defect of the ventricular septum.

Cramer and Fammel (*Arch. d. Mal. d. Coeur,* 16:561) report a very interesting case of congenital mitral stenosis with interauricular septal defect in a dwarf aged 41 who never showed cyanosis, not even just before death. Other congenital anomalies in this case were an abnormally short small intestine and a peculiar fissure in the right kidney. The right side of the heart was about twice as large as the left, the latter together with the aorta being quite hypoplasic.

Blair Stewart (*Jour. Lab. and Clin. Med.,* 8:454) also describes an instance of congenital cardiac disease in which congenital anomalies in other parts of the body were present, namely cleft palate and harelip. The heart showed a rudimentary atresic pulmonary artery, a common arterial trunk (*i.e.,* aorta) arising from the right ventricle, defective auricular and ventricular septa, patent ductus arteriosus, hypertrophy of the right heart and hypoplasia of the left heart. This case was four and one-half months old at the time of death following an operation for correction of the harelip. The child had paroxysmal attacks of cyanosis.

IX. HEART DISEASE IN PREGNANCY

The subject of heart disease in pregnancy has received considerable attention during the past few years. More exact diagnoses are being made of the nature of the heart symptoms and signs of which pregnant women complain, and the relation of these conditions to the course of the pregnancy are being estimated more accurately. Breed and White (Boston Medical and Surgical Journal, 188:984) analyzed the end-results in 102 cases referred to them for examination because of cardiac symptoms or signs. Forty-nine cases had symptoms of palpitation, tachycardia, non-progressive weakness or irritability, and the usual physical signs were a systolic murmur at the apex or base, ventricular extrasystoles or marked sinus arrhythmia. There were no diastolic murmurs and it was impossible to demonstrate in these cases any structural valvular change or any impairment in cardiac function. In the remaining 53 cases the lesions found were almost invariably of the rheumatic type, thus: mitral stenosis alone in 68 per cent., and with aortic regurgitation in 19 per cent.; mitral involvement without demonstrable stenosis in 9 per cent.; mitral stenosis with aortic regurgitation and stenosis in 2 per cent. and pure aortic regurgitation and stenosis in 2 per cent. Deaths occurred in two cases, one with acute endocarditis superimposed upon a chronic lesion and one with auricular fibrillation and mitral stenosis. From this study these investigators conclude that "approximately 50 per cent. of pregnant women who present cardiac symptoms or signs have no organic heart disease; second, that heart disease in pregnancy is almost invariably rheumatic in type; and third, that individual prognosis must be based upon functional capacity rather than structural change."

From his experience in the cardiac clinic of the Boston Lying-In Hospital B. E. Hamilton (Boston Medical and Surgical Journal, 188:987) has reached somewhat similar conclusions to those of Breed and White. Of all the cases in the hospital 2 per cent. have heart symptoms, and in only half of these can organic heart disease (almost invariably of the rheumatic type) be demonstrated; the remainder fall into the class of "effort syndrome" and allied conditions. Hamilton emphasized the need of more statistics and careful observations before the prognosis as to the behavior of the heart with organic disease in pregnant women can be more accurately estimated.

In Schaupp's series (*Cal. State Jour Med.,* 21:290) of 1395 pregnant patients seen in the Stanford Women's Clinic and the Stanford Obstetrical Service at the San Francisco Hospital, the diagnosis of cardiac disease (omitting functional murmurs) was made 38 times. The incidence in this group is higher than in that of Hamilton, who reports only 2 per cent., including both organic and non-organic cases. The great incidence of heart disease in Schaupp's series is the more striking from the fact that it is the general opinion of physicians who practice in California that rheumatic heart disease is less common on the Pacific than on the Atlantic Coast.

X. BLOOD PRESSURE

Bramwell, Hill and McSwiney (*Heart,* 10:233) measured the velocity of the pulse wave in man by means of a hot wire sphygmograph. making 74 observations in normal subjects of different ages. They found that during the interval between 10 and 60 years of age the volume-elasticity of the arteries is halved. This diminution in elasticity of the blood vessels has an important bearing on the increase in systolic pressure and pulse pressure with advancing age.

From a study of insurance company data from 1907 to 1919 Symonds (*Jour. Am. Med. Assn.,* 80:232) deduces that the systolic and diastolic pressures of both men and women increase with age and weight. In women the systolic and diastolic pressures are less than in men up to the age of 40; after this age the relation is reversed.

The existence of a clinical entity characterized fundamentally by raised blood pressure is further emphasized by numerous authors. From a study of the blood pressure in 15,000 university freshmen, Alvarez (*Arch. Int. Med.*, 32:17) found 18 to 26 per cent. of men and 3 per cent. of women with systolic blood pressures over 140 and suggests that this hypertension is hereditary and not acquired. Weitz (*Ztschr. f. Klin. Med.*, 96:151) investigated the families of 82 patients with so-called essential hypertension. He found that the parents of these cases died younger and more frequently of apoplexy or heart disease than those of comparable people with normal blood pressure. Ninety-three brothers and sisters of 42 cases of hypertension showed a higher incidence of hypertension than those of normal individuals. Hypertension in three generations is referred to. These observations seem to indicate a familial incidence of high blood pressure. Kylin (*Swenska läk-sallsk. Handl.*, 49:71) differentiates very strictly between hypertension of glomerular nephritis and essential hypertension. Agreeing with Volhard, he attributes the latter to disease of the vegetative nervous system, referring to the fact that adrenalin causes a lowering of blood pressure in essential hypertension, and considers this effect one of relaxation of the peripheral arterioles similar to the relaxation of the bronchioles in bronchial asthma. This author also reports a flattened blood sugar tolerance curve following adrenalin. He found no abnormalities of morphology or pressure in the peripheral capillaries in essential hypertension but a raised capillary pressure and irregularities in the capillary loops in glomerular nephritis with hypertension.

The relation between hypertension and its effect upon the heart is fully discussed by P. D. White (BOSTON MEDICAL AND SURGICAL JOURNAL, 189:1015). "Hypertension with or without nephritis causes cardiac hypertrophy unless it is slight or transient. Dilatation and congestive failure may ensue when the myocardium is sufficiently fatigued or when coronary arteriosclerosis or the toxic effects of infection or uremia are superimposed." Because of the existence of a combination of essential hypertension with heart disease, which is the direct result of this hypertension and with no real nephritis, this investigator strongly objects to the use of the term "cardio-renal," which was and often still is erroneously applied to this syndrome.

Strouse and Kelman (*Arch. Int. Med.*, 31:151) studied the effect of protein feeding up to 150 grams per day, and of the ingestion of strong stock soup and black coffee in cases of essential hypertension and found no change in blood pressure due to these factors. In cases of definite progressive nephritis with hypertension a diminution of protein intake sufficient to lower markedly the non-protein elements in the blood had no effect on blood pressure.

O'Hare and Walker (*Arch. Int. Med.*, 32:283) made careful observations on the effect of salt (NaCl) in the diet of patients with "vascular" hypertension and found it plays little or no part in this condition. They conclude that there is no reason for rigidly restricting salt in these cases.

XI. CAPILLARIES

The fundamental work of Krogh (The Morphology and Physiology of the Capillaries, Yale University Press, 1923), Müller and others has stimulated much active interest in the investigation of the morphology and physiology of capillaries under normal conditions and in disease. Hesinger (*Acta Med. Scandin.*, 58:31) studied 27 cases of pernicious anemia, and his findings may be summarized as follows: in severe cases it is difficult to see the capillaries because they are very pale red and the background is practically colorless; many capillaries are empty; in chlorosis and erythremia there are no such empty capillaries and the caliber is normal. In erythremia there is a relative increase in the number of capillaries.

In a brief communication to the Physiological Society of England, Thomas Lewis (*Jour. Physiol.*, 58: p. i) describes interesting observations on the force exerted by contracted capillaries. He measured the force required to cause a white line produced by stroking the skin of a normal individual to take on the tint of the surrounding skin. In one case the force required to distend the capillaries was about equal to the subject's systolic blood pressure, but this is exceptional as in most instances the 50 to 100 mm. of negative pressure required did not closely approach the systolic pressure. The observations reported help to explain why capillary distention does not take place in the foot on assuming the erect posture, since these capillaries are normally tonically contracted. Since the white reaction may be produced by slightly stretching the skin, it is suggested that the reaction comes into play in conditions of exercise, and that when, for example, a limb is moved the blood tends to be diverted from the skin vessels to the vessels of the muscles.

H. H. Dale (*Brit. Med. Jour.*, June 9 and 16, 1923, pp. 959 and 1006), in giving the Oliver Sharpey lectures on the activity of the capillary blood vessels and its relation to certain forms of toxemia, refers to the fact that histamin produces loss of tone in the capillaries, and that in shock there is a collection of blood in dilated peripheral capillaries. Thus there is some relation between shock and toxemias which cause peripheral stagnation by producing dilatation of the capillaries.

Ernst Boas (*N. Y. Med. Jour.*, 117:528) made observations on the capillary pressure in 47 patients with hypertension; 28 had normal or low capillary pressure (normal is the point at which the blood ceases to flow, due to mechanical pressure exerted—about 20 to 25 mm. mercury); 19 had a high capillary pressure. A year later 8 of the 19 were dead, while only 5 of the 28 had died. It would seem, therefore, that in such cases a high capillary pressure is suggestive of a poor prognosis.

XII. MURMURS

There exists, as yet, some difference of opinion as to the pathognomonic signs of mitral stenosis, particularly early slight stenosis. In a discussion of the interpretation of mitral and aortic diastolic murmurs, J. Edwin Wood, Jr., and Paul D. White (Med. Clin. N. Am., 7:729) state that in the presence of accessory signs such as accentuation of the pulmonic second sound, a snapping first sound and an electrocardiogram with marked right axis deviation and sometimes a high P wave, the rumbling mid-diastolic murmur may be considered pathognomonic of moderate mitral stenosis. They consider the crescendo murmur with presystolic accentuation a sign of marked mitral stenosis. On the other hand, the committee of the New York Association for the Prevention and Relief of Heart Disease defines the signs of mitral stenosis as either a mid-diastolic rumble in the presence of the typical systolic murmur of mitral regurgitation or this rumble followed by a crescendo murmur with presystolic accentuation. Judging from references to the signs of mitral stenosis found in the literature, not sufficient attention is paid to the mid-diastolic murmur; many authors still consider a presystolic murmur as a *sine qua non* to the diagnosis of mitral stenosis; this view is certainly incorrect.

Wood and White (*ibid.*) cite examples of cases in which mitral diastolic murmurs were heard and at autopsy the mitral valves were normal, in instances of adhesive pericarditis and cardiac hypertrophy and dilatation of unknown cause. A disturbed balance of the relation between the size of the dilated left ventricle and the normal mitral valve is suggested as the cause of this so-called functional diastolic murmur. They also refer to cases of aortic stenosis not detected clinically, and warn against attributing a loud rough systolic murmur at the base to roughening of the aorta.

Laubry and Daumer (*Bull. et Min. Soc. Med. d. Hop.*, 39:584) describe four cases in which a diastolic murmur typical of aortic insufficiency was heard and at autopsy the aortic valves were found to be normal. They attribute the presence of this murmur to dilatation of the left ventricle including the aortic ring.

XIII. ELECTROCARDIOGRAPHY

Every report of a relatively unusual electrocardiographic finding is in itself a definite contribution to this branch of the science of cardiology. This is not the occasion for a complete survey of all the important observations made during the year 1923, but a few will be mentioned.

Thomas Lewis (*Heart*, 9:283, 1922) reports the post-mortem findings in Starling's case of transient complete heart block. The patient had syncopal attacks during which there was cessation of ventricular beat. The fact that attacks were provoked by swallowing and could be temporarily abolished by atropin suggested that block was due to vagus stimulation. Two years later the patient died suddenly while working in a harvest field. The heart was enlarged and showed a fibrous mass under the bifurcation of the bundle, suggesting infarction due to embolism of a septal artery. This mass involved both branches of the bundle, these being lost in it. It is deduced that an abnormal vagal action cannot be regarded as primarily or wholly responsible for a patient's symptoms during transient heart block.

Willius (*Jour. Lab. and Clin. Med.*, 8:518) reports a case (the second on record) of ventricular fibrillation in a man, with temporary cardiac recovery. The patient had an attack of syncope lasting 10 seconds during electrocardiographic examination, then recovered and 34½ hours later died. Autopsy revealed an enlarged (610 gm.) dilated heart showing extensive fibrosis. The electrocardiogram of ventricular fibrillation showed irregular undulations following each other so closely that no period of cardiac inactivity is evident.

Wiltshire (*Heart*, 10:201) describes very interesting observations on the behavior of the auricles during periods of prolonged ventricular standstill. The patient had frequently recurring attacks of ventricular standstill during which he was unconscious. Continuous electrocardiographic tracings showed the following sequence of events: with the beginning of ventricular standstill the auricles at first are quickened, then begin to give evidence of exhaustion by a steady diminution of rate; with this there is a decrease in amplitude of deflection. Sooner or later the normal auricular complexes cease, irregular activity or irregular attempts at activity emanating from various foci appear. These are interspersed between attempts to establish regular slow rhythm, the stimuli for which may arise at the normal site or near it from some frankly ectopic focus. Finally there may be some periods during which the auricle is completely silent. On the resumption of ventricular activity, even though it be a single isolated contraction, there is a conspicuous and rapid recovery on the part of the auricle. Even when the auricles were in a state of flutter similar tendencies were seen. The flutter mechanism was not abolished but appeared to be progressively depressed, and like the normal mechanism showed rapid restoration as the immediate result of resumption of ventricular contraction.

Lewis and Drury (*Heart,* 10:179) report observations on the effect of vagal stimulation on intra-auricular block produced by cooling or pressure. They produced block in varying degrees between different portions of the auricular wall of the dog by applying pressure or cold. Stimulation of the vagus partly or entirely destroys this block, while the usual effect of depression on the A. V. node is obtained. Vagus stimulation improves conduction in auricular muscle fibers by reducing length of the refractory period, thus diminishing the block produced by pressure or cold. On the other hand, vagal stimulation exerts its usual effect and delays conduction in the A. V. node producing A-V block.

Hewlett (*Heart,* 10:9) describes a case which showed rapid ventricular rhythm with periods of auriculo-ventricular dissociation. The ventricular rate was controlled by a center near the A. V. node, for it was influenced by vagus stimulation and by atropin. Sometimes the ventricular rate was greater than the auricular.

Wilson and Finch (*Heart,* 10:275) report the interesting phenomenon of depression of the T wave of the electrocardiogram following the drinking of ice water. Normal individuals were given three glasses of ice water and the electrocardiogram taken with the subject in the recumbent position. Comparing tracings taken before and after the consumption of ice water, it was found that in the latter the T deflection was markedly lowered in lead II, inverted in lead III, and showed no change in lead I. The authors attribute this effect to cooling of the apex by the cold water in the fundus of the stomach with probable resulting delay in intraventricular conduction.

Ishihara and Nomura (*Heart,* 10:400) studied the behavior of the "spurious tendons" of the neuromuscular system in the isolated heart preparations of dogs and observed that the fibers contract rhythmically and synchronously with ventricular contractions in response to sinus control, and that they may contract independently if the bundle of His is severed; they also found that isolated fibers immersed in oxygenated Locke's solution at body temperature will continue to contract. These observations are in accordance with the belief that the neuromuscular system is made up of embryonic muscle fibers which have the power to contract independently.

Levine and Strong (*Heart* 10:125) report a case of paroxysmal tachycardia with irregularity of rhythm. The differences in time between beats varied from 0.01 to 0.19 second. They suggest that when irregularity is detected by auscultation during an attack of paroxysmal tachycardia, ventricular origin of the abnormal rhythm should be suspected since auricular tachycardia is regular. Marvin (*Heart,* 10:279) describes the interesting observation of gradual slowing of the rate in paroxysmal tachycardia (probably ventricular) from 172 to 124 per minute in the course of five days. In this case there was a curious febrile reaction to the tachycardia, vital capacity decreased progressively during the attack and there was no change in heart size.

The relative incidence of heart block in cases which reach the cardiologist may be judged from the figures given by White and Viko (*Am. Jour. Med. Sc.,* 165:659), who report an analysis of the cases of heart block found amongst 3219 patients. In this series there were 4 per cent. of the intraventricular and 4.8 per cent. of the auriculo-ventricular variety. Arteriosclerosis was the most common cause of both types and all degrees. Syphilis was present in 16 of 156 cases of auriculo-ventricular and in 11 of 130 of intraventricular block. Anti-luetic treatment, in the experience of these writers, did not affect the block. Partial auriculo-ventricular block is most commonly produced by digitalis, and more frequently in rheumatic heart disease than in arteriosclerotic heart disease. Digitalis, also, was responsible for six out of nine cases of sino-auricular block.

Less than 25 per cent. of the patients with complete heart block showed heart failure, either of the anginal or congestive type at the time of discovery of the heart block. Less than 25 per cent., also, gave a history of attacks of Stokes-Adams syndrome.

The mortality from heart failure during the past seven and one-half years was slightly higher in the intraventricular type than in the auriculo-ventricular. Heart failure (more commonly of the anginal type) occurred more frequently in the former than in the latter. Angina pectoris was associated four times as often with the former as with the latter. These observations with regard to the seriousness of the prognosis in cases of intra-ventricular heart block emphasize the value of electrocardiography, for this method is necessary for the diagnosis of such heart block.

XIV. VITAL CAPACITY

Peabody (*Northw. Med.,* 22:307) draws attention to the importance of vital capacity measurements. A diminished vital capacity is associated with heart disease accompanied by the symptom of dyspnea. This is explained by the fact that as one-third of the vital capacity is the depth of each respiration following exertion, a person with a diminished vital capacity must maintain the required minute volume of respiration by a greater increase in respiratory rate, hence dyspnea.

Lundsgaard and Scheerbeck (*Acta Med. Scand.,* 58:470) describe a more accurate method of measuring vital capacity by using the ratio between measurements of the size of the chest and various lung volumes in the given patient. They also take residual air into consideration. From a series of measurements on cases of rheumatic heart disease with mitral lesions they conclude that at first there is an increase in residual air, then with the development of congestion the total capacity of the lungs is diminished while the residual air continues to be more than normal. Finally, with more marked decompensation, the residual air also diminishes.

XV. X-RAY EXAMINATION OF THE HEART

A detailed description of the significance of the shape of the normal median cardiovascular shadow in the anterior view is given by S. Delhern and Robert Chaperon (*Jour. d. Radiol. et d'Elect.*, 7:1). This article does not lend itself to summarization but must be read entirely. It should serve as a good reference and so is mentioned here.

David S. Dann (*Arch. Int. Med.*, 31:269) made observations on the value of the index and angle of Bordet and Vaquez in cardiac examination (a method for measuring hypertrophy posteriorly) and found that only positive information obtained by this method is of any significance. Some normal cases showed "hypertrophy" and some with enlarged hearts appeared "normal" by this method.

According to Hodges (*Jour. Radiol.*, 4:351) the x-ray picture is not of any value in the diagnosis of pericardial effusion unless there is more than 200 c.c. in the pericardium. In the early stages of this condition the pericardial friction rub is of greatest significance.

XVI. MISCELLANEOUS

Newburgh and Clarkson (*Arch. Int. Med.*, 31:653) report well-controlled animal experiments in which typical arteriosclerosis resulted from feeding diets rich in protein. The occurrence and degree of arteriosclerosis was roughly proportional to the length of time the animal was fed with the high protein diet.

A case of severe cardiac insufficiency caused by isolated primary arteriosclerosis of the pulmonary arteries is reported by W. Movitz (*Deutsch. Arch. f. klin. Med.*, 142:115). The patient presented very severe acquired cyanosis, extreme hypertrophy of the right heart, and dilatation of the pulmonary arch without enlargement of the left auricle, observed in the roentgenogram. The lungs presented no abnormal signs.

J. K. Lewis and Hewlett (*Heart*, 10:1) observed that the vascular sounds (heard during blood pressure readings) are more striking after the injection of epinephrin, when there is an increase in pulse pressure rather than coincident with increase in rate.

It is of interest to physicians practicing in and about Boston that G. F. Strong and Albert Wilmaers (*Jour. Am. Med. Assn.*, 80:1038) tested the potency of a number of the commonly used digitalis and strophanthus preparations sold in this vicinity and found them up to the required standards in strength.

A number of interesting reports have been published on the effective use of intracardiac injections of adrenalin chloride in resuscitating the heart. P. B. Champlin (*Jour. Am. Med. Assn.*, 81:202), C. Bodon (*Lancet*, 204:586), E. Bauman (*Schweiz. med. Wchnschr.*, 53:198), J. Exalto (*Nederl. Tigdschr. u. Geneesk. Haarlem*, 67:1098) and others describe cases in which the heart beat had stopped and the injection of 0.5 to 10 c.c. of 1/1000 adrenalin resulted in immediate recovery of the heart. No reports of failures have been met with in the literature; these undoubtedly have occurred and will probably be published.

Kemal Djenad and A. Mouchet (*Bull. Acad. d. Med.*, 89:167) have observed in animals that when the beating heart is pricked with a needle there is an immediate temporary fall in blood pressure. This is a danger in a beating heart but not in one that has stopped, and this fact does not contraindicate adrenalin injection for purposes of resuscitation.

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Book Review

Selected Essays on Orthopaedic Surgery. From the Writings of Newton Melman Shaffer, M.D., F.A.C.S., Emeritus Professor of Orthopedic Surgery, Cornell University Medical College; Consulting Orthopaedic Surgeon, St. Luke's Hospital, New York, and to the New York State Orthopedic Hospital for Children, West Haverstraw. With Forewords by Dr. Lovett and Dr. Fisher, and Comments by Dr. Cotton, Dr. Nutt, Mr. Blagden, and Mr. Hardon. Illustrated. New York and London: G. P. Putnam's Sons, The Knickerbocker Press. 1923.

Dr. Newton Melman Shaffer, one of the best-known orthopedic surgeons of the country, has made many valuable contributions to the literature of bone and joint disease. In this volume of lectures and articles selected from his writings, his strongly held views as to etiology and treatment of many important morbid conditions are enunciated. While many surgeons will not always follow him, his contentions deserve the attention which a long and very active experience in the treatment of bone and joint disease merits. The papers cover a period of about thirty years, during all of which time the author has been actively engaged in practice. As an historical record alone, the collection is most valuable. Illustrations clarify the text. There are forewords by Dr. Robert W. Lovett and Dr. Edward D. Fisher and comments by other well-known surgeons.

Case Records of the Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY
RICHARD C. CABOT, M.D., AND HUGH CABOT, M.D.

F. M. PAINTER, ASSISTANT EDITOR

CASE 10121.

An American upholsterer of fifty-seven was sent from the Emergency Ward December 4 semicomatose, hard to arouse, and answering only in monosyllables or at random.

P. H. He had been drinking a good deal in the past year, especially in the past month.

P. I. Last week while drunk he was exposed to cold several times. Three days ago he began to vomit, and had fever, weakness and pain in his side and right chest. He rapidly became prostrated. The fever increased and delirium and stupor set in.

P. E. An obese, sick looking man breathing rapidly with slight expiratory grunt. Incontinent of urine. He was too ill for careful examination. He would not open his mouth or permit it to be opened. *Lungs.* No dullness or bronchial breathing. Numerous fine and medium crepitant expiratory râles at the right base. Apex impulse of the *heart* and midclavicular line not recorded. Left border of dullness 9 cm. Other percussion measurements normal. Rate rapid. Sounds of fair quality. *B.P.* 140/85. *Abdomen, extremities, pupils* and *reflexes* normal. *Genitals* not recorded.

T. 105.3°-101.4°-104.5°, always by rectum. *P.* 140-119. *R.* 36-44. *Urine.* Amount not recorded. Sp. gr. 1.020. A very slight trace of albumin, bile, 8-10 leucocytes per high power field and a few bile stained cellular casts. *Urine culture* negative. *Blood.* Hgb. 95%, leucocytes 15,000-14,000, polynuclears 70%, many young polynuclears, some with vacuoles in cytoplasm; slight anisocytosis. *Wassermann* negative. *Blood culture.* Streptococcus hemolyticus in both flasks. *Serum dilution* 1:125. *Non-protein nitrogen* 36 mgm.

Orders. December 4. Soft solid diet. Force fluids. Castor oil ℥ i every morning. Morphia gr. 1/8 s.c. every three hours p.r.n. for restlessness. (Not given.) Milk and molasses enema. Homatropin 2 drops in each eye. Turpentine stupes every four hours, follow with ice bag. Insert rectal tube while giving stupe. Castor oil ℥ iii every morning. Digitalis gr. iss t.i.d. Pillow to elevate left leg, and cradle over leg. Milk and molasses enema. 1 c.c. pituitrin intramuscularly.

The morning after admission there was a generalized icterus of the skin and sclerae. The patient responded better to questions. There was good result from the enemata, but not from the stupes. A few râles were now heard at the left base. The most urgent feature was marked abdominal distention. There was very slight pitting edema of the legs. On the left tibia were two areas of redness the size of a quarter dollar. There were varicosities. The visiting physician found some dullness, particularly in the right base, with diminished breath sounds; possibly high diaphragm; and on both sides slight changes in vocal fremitus. He found no enlargement of the heart to percussion.

December 6 the distention was considerably less. There were many fine and medium crackling râles at both bases. In addition to the red areas on the left leg there were red streaks extending up the inner aspect of the thigh for several inches, and a thrombosed vein extending up almost the entire length of the thigh. There was a red area on the dorsum of the left foot. December 7 the patient died.

DISCUSSION

BY DR. RICHARD C. CABOT

NOTES ON THE HISTORY

At a guess, this is the ordinary history of an alcoholic with pneumonia.

NOTES ON THE PHYSICAL EXAMINATION

"He would not open his mouth or permit it to be opened." Thereby we do not have the satisfaction of reporting as we do in every other case that he had pyorrhea, poor teeth, reddened throat, etc., which we could have a rubber stamp for just as well.

If this had been a thorough examination of his lungs the absence of dullness and bronchial breathing would be against the diagnosis I made. But when râles are localized, not distributed as in edema, they are almost as good evidence of pneumonia as if we had the evidences of solidification.

He ran a high continued fever, with the urine of infection and a very considerable jaundice. What is the highest serum dilution you have ever seen, Dr. Means?

DR. J. H. MEANS: Two to three hundred is a high dilution. Occasionally they go as high as eight hundred.

DR. CABOT: That is in intense jaundice cases. This patient had abdominal distention—one of the worst symptoms prognostically in pneumonia—which was obstinate.

DIFFERENTIAL DIAGNOSIS

As this case goes on I feel a great deal less inclined to a diagnosis of pneumonia. At the start there did not seem to be anything else that would account for so much dyspnea and edema. As we go on we get evidence of streptococcus septicemia, which is sufficient, even without anything in the lungs, to account for everything. As to the lungs, I should think the best guess is bronchopneumonia, not as the chief item but as one of the evidences of streptococcus septicemia, of which the blood is the best evidence, which is also shown by this phlebitis of one of the veins of the thigh.

I have seen cases just like this. I remember one vividly,—a man of almost the same age, with almost the same history, in which at post-mortem there was pus in every joint we opened. There were no special complaints of it. Were any of the joints looked at here?

Dr. Richardson: No.

Dr. Cabot: We may assume what we like then. We know the proneness of a streptococcus infection to attack the joints, and in the absence of any negative evidence we have a right to suppose that it has done so. Knowing that there is streptococcus in the blood, where else does it usually go? To the joints and to the serous membranes. Hence we are looking for streptococcus peritonitis. He had a lot of distention, and every now and then we fail in thinking of this as a consequent to pneumonia when a genuine peritonitis is really present. But here there is really no decisive evidence. Let us take the other serous membranes. We have no signs of fluid in the pleurae; but that does not exclude it. In the pericardium again we have no evidence, but we have to expect again that it may be affected. I think that is as far as I can go.

Dr. Aub, have you anything to add that has not got into the record?

Dr. J. C. Aub: I thought there was just a possibility of acute yellow atrophy, and I thought he had double bronchopneumonia.

Dr. Cabot: I had forgotten the jaundice. Many forms of sepsis show jaundice. So I think the natural thing, knowing he has streptococcus hemolyticus in the blood, is to say that his jaundice is of that type. You never had evidence of solidification, never got bronchial breathing?

Dr. Aub: Just a good many râles and some dullness. The heart showed nothing strikingly abnormal to account for these.

A Physician: Aren't the red spots and streaks on his legs good evidence of acute endocarditis?

Dr. Cabot: We have evidence of phlebitis in one vein; that is definitely stated. We know that one vein has that, and we know that lymphangitis starts with red areas. So it seems simpler to say lymphangitis. But it would not surprise me to find that acute endocarditis has started, though we have no other evidence of it.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Bronchopneumonia.
Septicemia.
Phlebitis.
Jaundice.

DR. RICHARD C. CABOT'S DIAGNOSIS

Streptococcus septicemia.
Bronchopneumonia?
Phlebitis.
Lymphangitis?
Icterus.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesions*

Septicemia, streptococcus hemolyticus.
Abscess of liver.

2. *Secondary or terminal lesions*

Icterus.
Infarcts of lungs.
Soft hyperplastic spleen.

3. *Historical landmarks*

Slightly patent foramen ovale.

Dr. Richardson: We were not permitted to examine the head or to open the vessel in the leg, but from the description one would think there was some phlebitis.

Dr. Cabot: Do you think your general permission would give you the right to put a needle such as we use for the pleura into the joints? Do you think that would be exceeding your permission?

Dr. Richardson: If anything turned up in criticism of the examination it would be said that I exceeded the permission. The permission to make the examination and the extent are written on the permit and I follow them.

Dr. Cabot: One doesn't want to do anything that would not be permitted. You think that would be contrary to the understanding?

Dr. Richardson: It depends entirely on the permission. In a case like this one would have to be very careful, because the organs were all replaced, and that indicates that we must follow carefully the incision restrictions. Of course the physician obtaining the permit may be able at times to have the restrictions modified.

There was distinct jaundice. The area described on the leg could be seen at the time of necropsy. On pressure along the leg and thigh

I could express no definite thrombotic clots from the upper end of the femoral vein.

The abdomen was distended, but the wall yielded. There was no fluid in the peritoneal cavity. The appendix presented in a position in which it is found once in a while. The cecum was well over in the median line just below the umbilicus, and the appendix lay a little to the left of the median line with its tip just above the pubis. The appendix was large and long, 8 cm. It was negative.

The intestines were negative except for a little reddening of the mucosa. The pleural cavities contained no fluid, and there were no pleural adhesions. There was much reddish mucous fluid in the trachea and bronchi, which were otherwise negative.

The tissue generally of each lung was pale to dark red, spongy, and yielded considerable fluid. In places there were blackish red areas, and in each lung there were a few infarcts. This condition of the lungs was associated with the streptococcus infection which was present. There was no pneumonia.

Dr. Means: Were these infarcts broken down at all?

Dr. Richardson: No. On one side, in the left lung, there was a place where some of the areas were a little boggy, but there was no definite suppuration.

Dr. Means: No abscess or gangrene?

Dr. Richardson: No. Of course some of those hemorrhagic infarcts might have broken down if he had lived longer.

The pericardium contained 200 c.c. of thin pale clear fluid, but was otherwise negative. The heart weighed 450 grams,—slightly enlarged. The myocardium was of good consistence, showing nothing macroscopically or microscopically. The walls of both left and right ventricles were thick—three to four mm. on the right, fifteen mm. on the left—the cavities full sized. The foramen ovale presented an opening four by two mm. The valve circumferences were large, but the valves were frankly negative. The coronaries were capacious, free and negative. The aorta and great branches were negative except for an anomaly; the renal arteries were given off from the aorta just above the bifurcation. At first sight I thought possibly we should find a horseshoe kidney, but we did not. The right kidney was in the usual situation but the left was down with its lower pole at the pelvic brim.

The liver weighed 2315 grams. That of course is a somewhat enlarged liver. (Normally 1200-2400.) The surfaces were smooth, the tissue punky and bile-stained. Continued sectioning disclosed in the right lobe, anterior portion, a short distance from the gall-bladder, an ovoid mass three by two cm. which turned out to be a solitary abscess in the liver. Cover-glass showed leucocytes and streptococci. The portal vein and radicles were negative.

The spleen was slightly enlarged,—246 grams—the tissue soft.

The kidney tissues were a little bile-stained.

There was a small hydrocele on each side.

The blood culture in life yielded the streptococcus hemolyticus, and cover-glass from the pus in the liver showed leucocytes and streptococci.

Dr. Cabot: Is there any section of the liver outside that place where the abscess was?

Dr. Richardson: Yes.

Dr. Cabot: Was there anything to explain this jaundice?

Dr. Richardson: The sepsis?

Dr. Cabot: They are always telling us that this "septic jaundice" is really due to changes in the liver. There is a good chance to follow it up. We are told that it is due to obstruction of the small bile-ducts in the liver.

Dr. Means: Jaundice is certainly very common in this type of infection, and in pneumonia also.

A Physician: Is there a hemolytic jaundice?

Dr. Cabot: If there is this is it. We used to think that it had something to do with changes going on in the blood stream, and hence to believe that there are two types of jaundice, that produced in the liver and that outside, theoretically the hemolytic type. Lately it has been believed that all jaundice is hepatogenous, coming from lesions of the liver. This is the first time I have had a chance to ask a pathologist how far that is verified.

Dr. Richardson: We have not identified it here.

Dr. Cabot: So that if you were asked the mechanism of this jaundice, aside from its association with streptococcus infection, you would depose and say, what?

Dr. Richardson: I should say I don't know, but that the hypothesis that there is some disturbance of the liver and ducts in icterus is a good one.

Dr. Cabot: Why should a change inside the cell give jaundice if it does not affect the bile-duct?

Dr. Richardson: It does affect the bile duct. The trend seems to be now to regard all icterus as due to bile duct obstruction of some kind or other.

Dr. Cabot: So that the sort of examination you made you would not regard as giving positive or negative evidence about that theory.

Dr. Richardson: It might or might not. The solution of this problem rests largely with experimental biologic chemistry.

A Physician: In these cases of so-called hemolytic jaundice does the urine ever show the chemical test for the presence of bile there?

Dr. Cabot: I do not see how the bile could develop in the blood stream without any function of the liver being affected.

If there is bile present in the urine it could not come from any changes taking place in the blood stream. It would have to come from the liver, wouldn't it? We do not know of bile being made anywhere except in the liver. That is what has driven people to the sort of investigation Dr. Richardson has spoken of, to see how that can be.

Dr. J. C. Aub: Cannot the blood pigments, the bilirubin and the like, be made outside the liver?

Dr. Richardson: Whipple and Hooper claim that bile pigments may be formed outside the liver, and not only that, but a change in diet increased the bile pigments, raising the question as to another source than hemoglobin for these pigments.

Dr. Aub: Wouldn't they appear in the urine?

Dr. Cabot: That isn't the usual view—is it—that we get any such thing as jaundice outside the liver?

Dr. Aub: I think Dr. Chester Jones also put the arm in very cold water and showed that the bile pigments increased in the plasma and that the hemoglobin broke down.

Dr. Cabot: Why was the liver so big, Dr. Richardson?

Dr. Richardson: Cloudy swelling and reaction to the abscess.

Dr. Cabot: Certainly we can say that hemolysis can go on without icterus. The question is, can it by itself produce the pigments that we call bile? I do not know enough to say that.

Dr. Chester M. Jones: Jaundice due to abnormal blood destruction certainly exists, and in such cases the yellow color is due to an increase in the bilirubin of the blood. We have definitely shown in cases of experimental intravascular hemolysis (paroxysmal hemoglobinurin) that hemoglobin could be changed to bilirubin in the blood vessels without the intervention of the liver. It is also true that jaundice can be due to toxic disturbances of the liver, with no evidence of obstruction to the flow of bile. In these cases there is undoubtedly functional damage to the liver parenchyma, with a resulting increase in the output of bile pigment. This increase is present both in the blood and bile. I should say that this case showed icterus on account of two, if not three factors,—(1) increased blood destruction due to a hemolytic streptococcus infection, (2) toxic damage to the liver due to a general septicemia, and possibly (3) some bile capillary obstruction.

CASE 10122

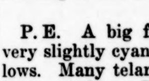
First entry. An American electrical engineer of fifty-eight entered September 26, complaining of dyspnea.

F. H. His wife had had two miscarriages, one she thought due to a fall, the other to lifting.

P. H. He had measles and whooping cough. At twenty-eight he was laid up for five months with sciatic rheumatism. Twenty years ago a lodge doctor found sugar in his urine, but admitted him to the lodge. He had no treatment. No doctor had found sugar since that time. For the past ten years he had had a somewhat prickling, burning sensation in the feet, usually relieved by putting his feet out from under the bed clothes. Occasionally he had the same sensation during the day after sitting still for some time. He drank a great deal of water. He urinated ten or twelve times a day. At night he urinated once or twice, only when nervous and walking about.

P. I. Two years before admission he had black stools with clots for three or four months. This cleared up under catharsis prescribed by a physician, and for six weeks he was free. Since that time he had occasionally had bright blood in the stools for two or three days, the last time a week before admission. A year ago he had high blood pressure and slight dizziness for a few minutes every three or four days, especially when excited at his work. Since changing to less responsible work nine months ago he had had no dizziness. A physician gave about fifteen drops of digitalis. For eight months he had had dyspnea on exertion and slightly productive cough with bloody sputum. The cough was at night only and lasted about six weeks. As part of his treatment some bad teeth were removed. He now coughed only after lying on his left side. The dyspnea first came on slight exertion and disappeared after a few moments' rest. Recently it had gradually increased. He had some palpitation with the dyspnea when excited or after exertion. He had very slight headache occasionally after hard work or excitement. For the past five nights the burning sensation in his feet had been worse. The night before admission he could get no relief and slept only an hour. That night he noticed for the first time swelling of the ankles.

P. E. A big framed, somewhat obese man, very slightly cyanotic, comfortable with two pillows. Many telangiectases. Xanthoma in each inner canthus. *Lungs.* Chronic passive congestion of the bases. *Heart.* Large. Apex impulse not recorded. First and second sounds replaced by systolic and diastolic murmurs at the apex and the base. Corrigan pulse. Artery walls palpable and tortuous. *B.P.* 160/40-230/35. *Electrocardiogram.* Normal rhythm. Inverted T_2. Rate 90. *Abdomen* large and soft. *Extremities.* Slight pitting edema. *Genitals.* Undescended right testicle. *Rectal examination.* Large cluster of external



hemorrhoids. *Pupils.* Left larger than right. Normal reactions. *Reflexes* normal.

T. 100°-97°. *P.* 100-61. *R.* 25-19. *Urine.* Normal amount, sp. gr. 1.009-1.016, the very slightest possible trace to a very slight trace of albumin at three of four examinations, leucocytes at all. *Renal function* 45%. *Blood* normal. *Non-protein nitrogen* 30 mgm. *Stool* negative. *Three Wassermanns* strongly positive. *South Medical consultant.* The kidneys being apparently in excellent condition this patient should be given either inunction or intramuscular injections of a soluble mercury salt for two weeks, then six or eight small intravenous injections of arsphenamin, these in turn to be followed by more mercury in some form.

Orders. September 26. Digitalis gr. ivss t.i.d. for two days. Fluids in moderation. Bed. Tannic acid and opium ointment applied locally to hemorrhoids once or twice daily. Sterile s.c.; if still awake and restless in an hour give codeia gr. 1/2 by mouth. (Given.) September 27. Sodium bromide gr. xx. September 28. Chloral hydrate gr. v in hot milk. September 29. Digitalis gr. iss daily. September 30. Digitalis gr. iss t.i.d. Pyramidon gr. v. October 1. Pyramidon gr. v at bedtime. October 3. KI five drops t.i.d. October 7. Heyden's solution 8 minims intramuscularly every other day. October 10. Pyramidon gr. v.

The patient did well. He complained of much pain in the legs at night, not relieved by various drugs. The history of blood in stools was believed to be explained by hemorrhoids, which he thought he had had all his life. The liver edge could be felt, but the liver was not definitely enlarged. October 13 he was discharged relieved.

History of interval. Three days after leaving the hospital he had a recurrence of epigastric discomfort occurring three times a day, sometimes before, sometimes after meals, not relieved by food, with nausea and vomiting of food just eaten about once daily after meals or even after drinking coffee or water. The abdominal complaint remained about the same until his reentry. He had been confined to the house. Ten days after leaving the hospital dyspnea recurred to about the same degree as before admission, but without corresponding constant orthopnea. There seemed to be some connection between the epigastric pain and the dyspnea. Both were more likely to occur when he lay down and were relieved by sitting up. Two weeks before readmission he noticed swelling of the feet, legs and "intestines." He had had constant anorexia since his discharge. His bowels were very constipated. The last movement was three days before readmission.

Second entry December 31, two months and a half after his discharge.

P. E. Moderately cyanotic, lying on the right side, dyspneic, with grunt. Ptosis of right eyelid. *Heart.* Sounds very distant. No murmurs made out. *B.P.* 175/40. *Lungs.* Abnormal but not recorded. *Abdomen* distended with gas. Questionable small amount of free fluid. Marked edema of abdominal wall, scrotum, penis, legs, thighs, over sacrum and back, more on the right side.

T. 99.9°-98.5°. *P.* 120-90. *R.* 30-24. *Urine.* Amount normal on the one occasion recorded, sp. gr. 1.018, a very slight trace of albumin. *Blood.* Hgb. 65%, leucocytes 13,000, polynuclears 84%, reds 4,320,000, achromia, considerable anisocytosis with several macrocytes and microcytes. Platelets slightly increased.

Orders. December 31. Digifolin gr. vi intramuscularly. Bed with head rest p.r.n. Karell diet. Digitalis gr. vi at 2 p.m., gr. iii at 6 p.m. Soapsuds enema. Later rectal tube with turpentine stupes. (Not given.) Caffein gr. vi s.c. p.r.n. Milk and molasses enema, if not relieved try pituitrin 1/2 c.c. s.c. and repeat in one-half hour. (Relieved very slightly.) Morphia gr. 1/6 s.c., repeat twice every three hours p.r.n. January 1. Pituitrin 1/2 c.c. s.c. Caffein sodium salicylate gr. vi. Liquids without milk, not over 60 ounces. Digitalis gr. vi twice to-day. Morphia gr. 1/6 s.c. immediately and every three hours p.r.n. January 2. Digitalis gr. iii t.i.d. to-day. Warm boric irrigation of right eye 2 i.d. Nitroglycerin gr. 1/100 by mouth for anginal attacks. Soapsuds enema. Atropin gr. 1/100 with morphia gr. 1/4 s.c. Bathe penis with warm boric solution b.i.d. with cleaning. Theocin gr. v t.i.d. for three days.

The patient had bad attacks of cardiac pain with severe dyspnea. The pain seemed to be in the epigastrium and was somewhat relieved by enemata, only slightly by morphia. January 3 he suddenly died.

DISCUSSION

BY DR. WILLIAM H. SMITH

NOTES ON THE HISTORY

It would be interesting to know a little more about this "sciatic rheumatism" at twenty-eight. At this age it is unusual. The questions arise, is it associated with some error in posture? is there an arthritis of the spine? or is there a hidden manifestation from some Neisser infection? could it be a manifestation of some toxic condition associated with glycosuria?

This sensation in the feet is one often complained of, and yet we find no pathology. Frequently it occurs in arteriosclerotics with hyperpressure, and may have something to do with the narrowing of the vessels in this disease. The sitting position, with pressure on a vessel, will frequently explain some of the unusual pains that occur in the thighs and legs in elderly people of arteriosclerotic age. It is probably allied to the pain experienced on exertion in the legs, and is called angina cruris. I remember one case with many orthopedic opinions. The actual diagnosis was obtained from one fact in the history; he could sit in his office all day without discomfort or pain, and five minutes seated in the Pullman car would produce pain. I asked him how deep his office chair was, and he said about eighteen inches. The depth of the Pullman chair estimated brought pressure on his popliteals. Working on this basis of pressure and putting him on nitroglycerin, he has been perfectly comfortable since.

Polyuria is a valuable symptom. Polyuria based on a large liquid intake in a nervous person has no value. A person who drinks abundantly of water or milk and is wakeful during the night frequently has polyuria; it is of no consequence.

It is a bold physician who prescribes catharsis in the presence of black stools and clots.

Here of course one thinks of the possibility of cirrhosis of the liver or duodenal ulcer.

The fresh blood in the stools is to be explained by his hemorrhoids.

There is no question if we take a man with high blood pressure and teach him to ease up in his method of life, shift his responsibility, we shall do a good deal for that man. Someone evidently gave this patient very wise advice.

(Fifteen drops of digitalis.) It is better to prescribe digitalis in liquid form in the accurate minim glass measure.

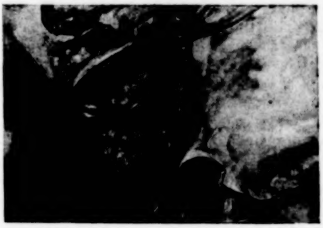
In other words his failure of compensation had been progressive. In the beginning compensation easily restored, now a progressive failure of compensation.

In regard to the sensations complained of in the feet and legs I have found that nitroglycerin or sodium nitrite works very well where the diastolic pressure is high. Oftentimes this sensation in the legs is the chief complaint of these patients,—if they could only escape from the sensation in the feet and legs. It is quite striking.

NOTES ON THE PHYSICAL EXAMINATION

This is a bad type of machine for a cardiac case. I see many patients with cardiac disease with a history of a year or two of failure of compensation, and during this time they have been permitted to carry fifty, sixty or even seventy pounds of extra weight. More attention should be paid to weight reduction in the obese cardiac case than at present appears to me to be paid.

Of course the master key in the diagnosis in this case should be the absence of the aortic second sound. Why is the aortic second absent? The aortic second is absent where there is aortic stenosis with fixation of the valve, in which case we have usually a limitation of the aortic regurgitation. (See illustration.) It is absent where we have marked retraction of the aortic valves against the aorta as in syphilitic aortitis. The column of blood in the aorta, with no support at its base, the valves being held up against the aorta by the syphilitic process, makes extensive regurgitation of the aortic valve probable. It is not limited as in the case of aortic stenosis. In this type of case, the syphilitic aortitic case, there is a corresponding high pulse pressure and extreme cardiac hypertrophy when the syphilitic aortitis is low in position and the valve retraction is extensive.



Extensive arteriosclerosis of the aortic valve; stenosis, with limited regurgitation. A little less than natural size.

After a water color painting by H. F. Aitken.

DR. WILLIAM H. SMITH.

The aortic second is sometimes absent in extensive arteriosclerosis with calcification of the valve. The three conditions in which I associate absence of the aortic second are:

(1) A complete chronic infectious process—aortic stenosis with limited regurgitation.

(2) A complete syphilitic aortitic process with valves retracted closely against the aorta.

(3) A complete arteriosclerotic process with fibrous or fibrocalcareous fixation of the valve.

The T-wave may have been an effect of the digitalis.

The hemorrhoids probably explain the recent fresh blood in the stools. One could not interpret his hyperpressure on a chronic nephritis background with the evidence as here presented.

Of course the palpable liver edge can be explained by his passive congestion, or one might speak in terms of cirrhosis, associating with the cirrhosis the loss of blood and dark stools which he had one or two years before.

Whether his epigastric discomfort should be interpreted in the terms of drugs—digitalis—passive congestion of the liver, angina pectoris, or the pain of a duodenal ulcer, I do not see how one can decide on the evidence at present.

With the underlying cardiac pathology, low aortitis, valve retraction, the position of the pathological lesion is correct for angina pectoris.

A cardiac condition which responds to treatment such as this man had here, with symptoms recurring so shortly afterward, when the patient has not apparently been careless, suggests a cardiac condition which is of the utmost gravity. In other words there is a condition mechanical or complete which renders it impossible to compensate the underlying pathological condition.

If one takes the history of mitral stenosis, associated with aortic stenosis, with or without tricuspid stenosis, we shall find there a history of progressive failure over months. When this regular shrinkage in the field of the cardiac response appears in the history it is exceptional when we accomplish much by rest, digitalis or drug therapy, and the pathological findings at the necropsy table of marked stenosis and completed process are the rule. Such histories are also to be obtained in syphilitic aortitis when low, and in the dying myocardium due to arteriosclerosis. This patient gained somewhat in compensation because his orthopnea was relieved; his dyspnea, however, continued and progressed.

Of course it would be interesting to know a little more about the type of pain here presented, whether it were an angina pectoris. Angina pectoris breeds pretty true in symptomatology if we know how to ask our questions. Continuous angina is very suggestive of coronary occlusion. Coronary occlusion makes possible the erection of the thrombus on the inner surface of the left ventricle, and we can have embolic infarction simulating a cerebral hemorrhage, or even emboli from the mural thrombus may produce acute abdominal symptoms puzzling alike to the internist and the surgeon.

Here we have the history of ulcer possibly, the history of angina probably, and no means of differentiation so far as I can see. The description of this man's pain is not characteristic of angina unless of mechanical angina. It is conceivable, and we have had one case where a polypoid mass on the aortic valve was placed so that intermittent angina could be explained.

In other words a patient coming in with a systolic and diastolic murmur at the apex and base replacing the heart sounds, at his first entrance, now comes in with no murmurs heard and sounds very distant. One can interpret that in terms of failure of compensation or in terms of dilatation of the heart or hydropericardium. Certainly the murmurs are not absent because of low blood pressure with a systolic of 175 and a diastolic of 40. This high pulse pressure, taken in connection with the absence of the aortic second, makes extensive aortic regurgitation usual. The edema furnishes more evidence of failure of compensation, if it is needed. There was a temperature ranging from 100° to 97° before at entrance.

At this urine examination the gravity is higher than either of the gravities recorded before, and again makes us feel that the kidney as such—a chronic nephritis to explain the high pressure—is out of the picture.

There is no marked change in the blood picture,—leucocytosis and secondary anemia. The anemia is somewhat similar to a primary anemia, but with an increased platelet count, which is unlike primary anemia, more like secondary infection.

I do not know how to make a diagnosis of the case. If we use the master key, the absence of the aortic second, the high pulse pressure and the very large heart, the best diagnosis would be syphilitic aortitis—low—with extensive valve retraction. Being low in position, near the coronaries, it would explain the pain in terms of angina pectoris. I do not see as we follow this case along how arteriosclerosis could explain the entire picture. It is possible that a very extensive polypoid vegetative endocarditis of the aortic valve impinging on the coronaries might produce the attacks of angina.

The large heart, the extensive degree of regurgitation through the aortic valve, are more consistent with syphilis of the arch. The age of the man, the history, the high pressure, the systolic and diastolic murmurs, the absence of the aortic second sound, the marked enlargement of the heart, the attack of angina, the three positive Wassermanns, and the type of cardiac death are consistent with syphilitic aortitis.

Suggesting a fresh vegetative endocarditis of the aortic valve are the leucocytosis, temperature, and variations in the murmurs. Less is to be explained on the syphilitic aortitic diagnosis than on the polypoid vegetative endocarditis of the aortic valve.

A PHYSICIAN: Was there any x-ray evidence?

DR. SMITH: There was none given.

DR. WILLIAM D. SMITH: We do not know that it is syphilitic aortitis.

There is one thing that particularly impresses me in this case, and that is the very high blood pressure. In the cases of syphilitic aortitis that I have seen, while they may have had high blood pressure with regurgitation, they have not, generally speaking, run a very high systolic pressure. And if I see a case with as high a pressure as that I am a little prejudiced against its being a syphilitic case. Yet every once in a while we do see the two things together. But I am a little inclined to think it is coincidence rather than cause and effect. I do not believe that syphilis, even a vascular syphilis, gives a hypertension of that degree. Most of the cases I have seen have not had such a high pressure even before decompensation.

On the assumption that this is a syphilitic case with aortitis and death Dr. Cabot asked me to say something about the use of arsphenamin in these cases. It is very hard to evaluate the standing that arsphenamin has. It is easy enough to get many cases in which the use of arsphenamin right away, without preliminary treatment by mercury and iodid, has given excellent results. The most striking cure of an angina that I ever saw was in a man of seventy who for a month had intense paroxysms. He was given at once relatively large doses of arsphenamin with no discomfort and no reaction, and with extraordinarily good effects; a year or two ago that man was alive and doing some work. On the other hand it is just as easy to find cases which have done badly when they have been given treatment with arsphenamin. About a year ago I saw a man of thirty-eight who had no history of syphilis. He did have an aortitis, an enlarged heart; he had had no decompensation, no cough, no shortness of breath. The x-ray showed a slight dilatation of the aorta. That man got three-tenths with essentially no preliminary treatment, with no immediate reaction. Two days later he was sitting comfortably in his chair and dropped dead. Granting the likelihood of the aortic regurgitation case to sudden death, we should have expected him not to die for a long time. He had a relatively good prognosis. I think different management might have had a different effect on the patient.

Wile, at Ann Arbor, has expressed the opinion that arsphenamin is not indicated as a routine procedure in central vascular syphilis, but that it may be used, and used with reasonable safety, in selected cases that have previously been given mercury and iodid. Two or three years ago Dr. Stokes of the Mayo Clinic published several cases in which the use of arsphenamin had been followed by unfortunate consequences. I am going to read two or three of the quotations, because he says it very much better than I can.

In regard to a case of aortic aneurism with collapse and death after the first injection: "Arsphenamin makes a dangerous beginning for the treatment of late syphilis."

Again, in respect to a case of cardiovascular syphilis and neurosyphilis with death under treatment: "Arsphenamin, because of the therapeutic shock, because of the rapidity of the healing changes which it produces, and because of the vasculo-toxicity, is dangerous to bad hearts."

In a case of early diagnosis of syphilis of the heart with angina: "The treatment should be begun with mercurial preparations and subsequently arsphenamin and mercury should be pushed with vigor. We have seen much evidence that healing changes actually take place in the cardiovascular system which if too rapid give rise to severe complications."

I think that is probably the answer. The characteristic of arsenic preparations which is generally appreciated in the treatment of early syphilis, but is not thought so much about in the late visceral syphilis, is its rapid and fulminating effect. It is followed by extraordinary absorption of gummatous tissue and syphilitic infiltrates, and obviously that is an advantage in early syphilis. While I am not qualified to discuss in any detail the pathology of the lesions or the tissue changes under treatment, broadly speaking the result is the absorption of syphilitic infiltrations with the formation of scar tissue, and it is reasonable to suppose that such a vital organ as the heart or aorta might be better preserved by slow than by rapid treatment.

My own impressions are about as follows: I think arsphenamin is a useful drug and should be used in the treatment of central vascular syphilis. I think mercury and iodid are the indispensable drugs. I think treatment should always be begun with mercury and iodid for several weeks. Then I think it is permissible to use arsphenamin, but I think the dose should be pretty small to start with, not over one-tenth. I believe that to institute treatment with arsphenamin on these cases is dangerous, and that the danger is proportionate to the size of the dose and the anatomical damage in the heart and aorta.

We find plenty of physicians who start these cases on arsphenamin and get away with it. But it is my feeling that it should not be used as preliminary treatment. Furthermore, I have become very skeptical of the effect of arsphenamin when definite and reasonable treatment with mercury and iodid has not relieved symptoms or accomplished anything. And of course it is the relief of symptoms that we get. We do not change the condition, but we often strikingly relieve the symptoms, and we may slow down the process, stop the active syphilitic process that is going on in the tissue. That is what we hope to do.

A PHYSICIAN: I have often wondered what the evidence was that these lesions of the central vascular apparatus were recent. Some of them look as if they might have been there for some time.

DR. WILLIAM D. SMITH: I do not think they are recent, but there is a difference whether we find a slight dilatation of the arch and a slight murmur or a huge heart and a tremendous murmur in which there have already been signs of decompensation. Of course the spirochetes are colonized there very early in infection and lie latent for a while. Around the lymph spaces of the first portion of the aorta is a favorite place, and they stay there. I do not suppose they are ever "early" from the point of view of microscopic examination. Of course when we get a man, no matter how little damage or change there may be in his aorta or heart, it is not a recent infection; it has been there, but we could not find it. Often it can be found by fluoroscope or x-ray when certainly the average doctor cannot find it by percussion.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Luetic heart disease.
Aortic regurgitation.
Congestive failure.

DR. WILLIAM H. SMITH'S DIAGNOSIS

Syphilitic aortitis (?)
Aortic regurgitation.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesions*

Luetic aortitis with involvement of aortic valve.

2. *Secondary or terminal lesions*

Arteriosclerosis.
Hypertrophy and dilatation of heart.
Chronic passive congestion.
Infarcts of lungs.
Hydrothorax.
Hydropericardium.
Ascites.
Anasarca.
Compression atelectasis of lower lobe of right lung.
Septicemia, streptococcus.

3. *Historical landmarks*

Obsolete tuberculosis of a bronchial lymphatic gland.

Dr. Richardson: There was a moderate amount of subcutaneous fat. We were not permitted to examine the head. There was marked edema of the scrotum, the penis, and the feet and legs, and in the region of the right elbow there was swelling and pitting. The face and lips were dusky purplish, the skin elsewhere rather pale. The abdomen was slightly distended but the wall yielded.

The subcutaneous tissues were wet and dusky, the muscles large and negative. In the peritoneal cavity we found 1000 c.c. of pale clear fluid. The appendix and esophagus were negative. The stomach and intestines generally showed dark red velvety mucosae oozing thin bloody fluid,—a typical picture of chronic passive congestion. The anterior margin of the liver was ten cm. below the costal border.

In the right pleural cavity there was 2500 c.c. of thin pale clear fluid, in the left 1000 c.c. There was compression atelectasis of the lower half of the right lung, not so marked on the other side, where the fluid content was much less. There was a little calcareous degeneration of one of the bronchial lymphatic glands. The lungs showed typical chronic passive congestion and in places infarcts.

The pericardium showed 100 c.c. of thin pale clear fluid, a slight excess. The membrane otherwise was in good condition. The heart weighed 650 grams,—considerably enlarged. The myocardium was of good consistence, bluish-brown-red. This bluish-brown-red color is indicative of congestion. The right ventricle measured four mm., the left 13 mm.,—thick walls. There was considerable dilatation on the left, especially in the region of the left ventricle, which was more or less basin-shaped. This kind of cavity is associated with syphilitic aortitis more than with any other lesion. There was considerable dilatation on the right, and the cavities all contained much currant-jelly-like blood clot, as if the heart had stopped distended. The mitral valve measured 11 cm., the aortic 6.5 cm., the tricuspid 13.5 cm., the pulmonary 8 cm. There were changes on the aortic valve. The other valves were negative.

A process on the aorta extended down along the posterior walls of the sinuses of Valsalva and bound the contiguous margins of the aortic cusps down by broad fibrous bands and with a moderate amount of fibrous thickening of the cusps with decrease of their widths,—the picture of course of an involved aortic valve in syphilitic aortitis. The same process extended around the coronary orifices, but did not occlude them,—decreased the orifices slightly. The coronaries beyond their orifices were free and showed a slight amount of fibrous sclerosis.

The aorta: the ascending thoracic portion, 8 cm. That is enlarged, of course. The descending portion 7 cm., enlarged, and the abdominal portion of full size. Extending from the walls of the sinuses of Valsalva up the ascending thoracic was a process which all told resembled quite well that of syphilitic aortitis. In addition to this there was more or less arteriosclerosis.

The liver showed typical nutmeg markings. The spleen showed chronic passive congestion.

The kidneys weighed 345 grams, the tissue plump, bluish-brown-red, microscopically and macroscopically showing typical chronic passive congestion.

Culture from the heart blood yielded a slight growth of streptococcus, that is, a terminal infection.

Dr. W. D. Smith: Do you ordinarily associate extreme hypertension with the luetic disease?

Dr. W. H. Smith: No, not necessarily.

I think there is one thing you do not emphasize as much as I have been taught to clinically; that is low aortitis where there is valve retraction and coronary impingement. The history of angina, with possibility of edema around the coronaries,—it seems to me that is the great crux, if you choose. High aortitis is one thing;

low, with valve retraction and coronary impingement, is another.

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CASE 10123

An Irish contractor of forty-eight entered December 7.

F. H. Not obtained.

Habits. He had always used alcohol rather freely.

P. H. He had always been well except for an illness lasting six weeks twenty-five years ago, presumably typhoid fever. He had had a few attacks of "indigestion."

P. I. For the past year he had had double inguinal hernia, much larger on the left, and had worn a truss. Both herniae were always easily reduced. That on the left had frequently been painful. Ten days before admission the left hernia became more than usually painful while down in its sac, but was easily reduced. The reduction was followed by general abdominal discomfort and dull pain in the left lower quadrant. He felt feverish and sick. The pain had continued and the abdominal discomfort had increased. December 2 a doctor found his temperature 102° and made a diagnosis of possible typhoid fever. In spite of catharsis and enemata he had had only one small watery movement in ten days, after a dose of salts six days ago. During the past three days the abdominal discomfort had increased.

P. E. A well nourished workingman with flushed cheeks and somewhat labored breathing. Skin hot and dry. Teeth carious. Alcoholic breath. Tongue dry, with brown coat. *Heart* not enlarged. Action rapid, regular, sounds of fair quality, no murmurs. *Lungs.* Some emphysema in both chests. No râles or dullness. Slight hyperresonance above the normal lung base line. *Abdomen* distended Wall felt boardlike throughout. Marked tenderness in both flanks accentuated just above the pubic on the left. Right inguinal canal free. Internal ring admitted one finger. Left canal free. Internal ring filled with soft tissue—omentum (?). A hard mass, moderately tender, high up on the left could just be reached by the finger tip. *Genitals, extremities, pupils* and *reflexes* normal.

Before operation *T.* 100.2°, *P.* and *R.* not recorded, *leucocytes* 15,000.

Operation was done at once. The patient was put on the dangerous list immediately after it. On arrival in the ward he was placed in extreme Fowler position with constant rectal seepage. Next day he seemed very bright and in surprisingly good condition. He was given strychnia and brandy at frequent intervals. That night he was given a calomel order with very slight result. December 9 his pulse became more rapid, he was mildly delirious, grew steadily worse, and died in the afternoon.

DISCUSSION

BY DR. EDWARD L. YOUNG, JR.

Attacks of pain in a hernia are always symptoms that make one advise a patient to have something done about it, because they are often due to a slight pinching of the contents of the sac, and mean that that hernia could get into trouble very easily.

This account at once excites suspicion of reduction *en bloc.*

We note from the number of this necropsy that it was back in the days when typhoid fever was fairly common.

As the story is given here it is very suggestive of a reduction *en bloc,* that is, the internal ring still gripping the contents of the sac although the mass had disappeared from the surface on attempt at reduction. With that is acute intestinal obstruction and by this time probably a peritonitis.

It seems to me that this is a pretty clear-cut description of what we have spoken of, and the only question is whether he has been able to go ten days with a bit of strangulated gut in the sac, or whether it is just a bit of omentum and because of the caught omentum a kink of the gut with obstruction and no strangulation. Because strangulated hernia means strangulated gut, and that means gangrene in a short time with spreading peritonitis. From the description he has that. This is a healthy man and fairly young, so he may have gone with a gangrenous gut and beginning peritonitis for ten days.

Of course the only thing is to give him what chance there is with an operation under local—give him the quickest operation there is,—and the only thing he possibly would stand is freeing of the gangrenous gut and freeing of the omentum if it is there. At this stage of the game of course it is a very slender chance.

DR. YOUNG'S PRE-OPERATIVE DIAGNOSIS

Strangulated hernia.
Intestinal obstruction.
General peritonitis.

OPERATION

No pre-operative diagnosis is recorded.—Gas and ether. Five inch median incision below the umbilicus. The peritoneum was opened with the escape of yellowish cloudy fluid. A culture taken at this time showed a very slight growth of colon-like bacilli. The ileum was found markedly dilated, injected, covered with patches of fibrin, and in places black and gangrenous. Much pus was found free in the pelvis. On careful examination a ruptured gangrenous appendix was found. It was removed. One cigarette drain was placed to the stump. The patient was sent to the ward in fair condition.

PATHOLOGICAL REPORT

An appendix 5 cm. long, gangrenous and perforated.

FURTHER DISCUSSION

It never entered my head that he had an appendix. We had to say peritonitis. Of course he had peritonitis, and the outcome is the same, —the prognosis based on the peritonitis.

I assume he died of sepsis. I do not see how I can make a diagnosis of appendix now. I should like to know from Dr. Richardson what this is that they felt.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Appendix abscess
General peritonitis.

DR. EDWARD L. YOUNG'S DIAGNOSIS

General peritonitis.

ANATOMICAL DIAGNOSIS

(Gangrenous appendicitis with perforation.)
General peritonitis.
Operation wound, appendectomy.

Dr. Richardson: The appendix was absent and there was a general peritonitis. The organs in the abdominal cavity otherwise were negative. We were permitted to go in only through the operation wound in the abdominal wall, but the thoracic organs to the touch gave no evidence of pneumonia, and all we found was the peritonitis and the sutures at the base of the appendix.

Dr. Young: There was no hernia at all?

Dr. Richardson: None noted.

INSTRUCTION IN NUTRITION

A second nutrition institute was held at Indianapolis from February 4 to the 16th, which was of especial significance from the standpoint of the responsibility of school authorities for the nutritional condition of children. After eighteen months of experimentation following the first institute in this city, it is now planned to require this training course of all school physicians, school nurses and principals. At this meeting one elementary teacher from each school was also included in the group taking the work. The problems that arise in the division of the field between the Board of Health and the Board of Education have been met most successfully by a very effective plan of coöperation between the two boards.

On February 28, Dr. William R. P. Emerson spoke before the Department of Superintendents of the National Education Association at Chicago on "Physical and Mental Unfitness in Children."

The report for the last quarter in the Rochester schools shows that sixteen nutrition classes have made an average gain of 343 per cent. of what would be expected of normal children of the same ages in this period. This is an advance of 38 per cent. over what was accomplished in the corresponding quarter of last year.

From February 25 to March 8 a second institute was held in Newark, N. J. A special feature of this meeting was a series of lectures and demonstrations by Dr. Douglas A. Thom, in which he presented the work of his habit clinics. Dr. Joseph Eby of New York City spoke on "Malnutrition and Orthodontia," and Dr. Haven Emerson presented "The Relation of Malnutrition to Public Health Work."

Nutrition classes have recently been organized at Haverhill and in connection with the Plymouth Cordage Company at Plymouth.

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Week's Death Rate in Boston.—During the week ending March 8, 1924, the number of deaths reported was 240 against 283 last year, with a rate of 16.11 against 19.15 last year. There were 24 deaths under one year of age against 51 last year. The number of cases of principal reportable diseases were: Diphtheria, 40; scarlet fever, 148; measles, 181; whooping cough, 9; typhoid fever, 5; tuberculosis, 32. Included in the above, were the following cases of non-residents: Diphtheria, 2; scarlet fever, 11; measles, 1; typhoid fever, 1; tuberculosis, 8. Total deaths from these diseases were: Diphtheria, 2; scarlet fever, 1; measles, 2; typhoid fever, 1; tuberculosis, 19. Included in the above, were the following cases of non-residents: Tuberculosis, 5.

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The Journal does not hold itself responsible for statements made by any contributor.

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DR. CHARLES WILLIAM ELIOT

Charles William Eliot was born on March 20, 1834. By inheritance and selection he has belonged to Harvard. His father was Treasurer of the College, his grandfather founded the Chair of Greek Literature, and his uncle, the father of Charles Eliot Norton, at one time held the Chair of Sacred Literature.

Graduating from Harvard, Mr. Eliot was for some time Assistant Professor of Mathematics and Chemistry, later assuming the head of the Department of Chemistry in the Scientific School. Resigning this post, ten years after graduation, he spent two years in the study of chemistry in Europe, returning to America to fill the Chair of Chemistry in the Massachusetts Institute of Technology. In September, 1868, the President of Harvard retired and Mr. Eliot was appointed his successor, assuming his position on May 19, 1869, at the age of 35 years.

Calvin Ellis had been appointed Dean of the Medical School in the same year, and in 1870, under the influence of these leaders, the reformation of medical teaching in this country was begun at Harvard Medical School, and the university was launched on its period of greatest development and prosperity.

Great as he is in many ways, we perhaps best appreciate Dr. Eliot's great services to medical education, and they have been appreciated in the past, for not only does he hold the honorary degree of Doctor of Medicine, but was this year elected Honorary Fellow of the Massachusetts Medical Society, whereby the society did honor to itself.

Bryce has said, "Neither the German Pro-Rector, nor the Vice-Chancellor in Oxford and Cambridge, nor the Principal in a Scottish University, nor the Provost of Trinity College in Dublin, nor the head in one of the colleges in Oxford or Cambridge, is anything like so important a personage in respect of his office, whatever influence his individual gifts may give him, as an American College President." In this selected group Dr. Eliot holds first rank, not only as the President Emeritus of the first university on the American continent, but by virtue of his own great qualities of leadership.

The dean among college presidents, a pioneer in educational reform, Dr. Eliot has aptly been termed the foremost private citizen in America.

TO CHARLES W. ELIOT ON HIS NINETIETH BIRTHDAY, MARCH 20, 1924

To you, sir, greetings we would bring today;
To you, devotion and affection give;
To you, our homage and our reverence pay,
Whose ninety years have taught us how to live.

As Fellow in the healing art we know
No man whose worth has greater been than yours;
No man who has so far increased the flow
Of every draught that Panacea pours.

A College in New England to your care
Was given for the molding of our youth.
In honoring you, men turn their steps to where
A University stands pledged to truth.

A humble School of Medicine of brick
Stood where a famous hospital now stands;
You visioned service for the people's sick,
And brick has turned to marble in your hands.

To Medicine of yesterday you taught
The creed that makes our Medicine today;
That glimpse of future service that you brought
Rewards in greater service still will pay.

Let us, physicians, render thanks to you;
To you who have so truly shown the road.
The task you started we will carry through,
Your code of service taking as our code.

Jog.

MEETING OF THE ASSOCIATION OF AMERICAN MEDICAL COLLEGES

The thirty-fourth annual meeting of the Association of American Medical Colleges was held at Omaha, Neb., on February 28 and 29 and March 1. The attendance set a record both in the number of institutions represented and in the number of delegates, as some schools sent several representatives. The next meeting will be held in Boston.

Correlation of subjects in the medical curriculum is receiving considerable attention, and reports were made of courses now carried on, in which special efforts are made to correlate "preclinical" and "clinical" experience. At St. Louis University a fairly comprehensive course in "Clinical Physiology" is given. The unit is a conference at which a student presents a brief paper, prepared under supervision and with assistance, on a given topic. Patients are shown illustrating the subject. There is discussion by the clinician as well as by representatives of the teaching staff of the preclinical departments. At the University of Pennsylvania for several years medical clinics have been held for the first year's classes. Here a single instructor conducts the course. In other institutions courses along similar lines are now undertaken.

The preliminary reports indicate no reason for discontinuing the experiments, and the interest taken by the students justifies further investigations along these lines. It is of course true that the student must do his own correlating as well as the rest of his thinking, and that there is danger of "spoonfeeding." But there exists a very definite and serious problem for which no generally recognized solution is at hand. It may be that the solution is to be found along the lines in which experiments are now being made.

The interne problem arouses a good deal of interest. It seems unwise for an educational institution to require, as a preliminary to its granting a certificate, work over which it has as little control as in non-university hospitals. Yet most graduates take a year of interneship, so requirement by the State would work little hardship under present conditions.

The number of interneships in approved hospitals is not quite as great as the number of graduates, the actual shortage in approved hospitals being due to graduates going to hospitals which are not approved.

There should be some standardization of interneship. The rotating service of one year, chiefly medicine and surgery, is widely favored. But for graduates who are going to specialize, non-rotating services of an additional year or more, leading to residentship, should be provided. It must not be forgotten, however, that the formal requirement is not as important as the actual training received, a personal matter, depending on the quality of the staff.

In the past twenty years there has been great progress in medical education, in large part due to standardization. But one of the results of the rigidity of standards which has ensued is that medical education is in the grip of the States, rather than in the hands of strictly educational institutions. This condition acts as a drag on progress.

It is not clear just how the remedy is to come, but we are confronted with fundamental educational problems, in the solution of which the best pedagogical talent and experience are needed. It is not clear, for example, what the two years of premedical work in college are to represent. If they represent preparation for the medical studies should they not be determined in content and method by the medical school? If they are to give a general cultural background, should they not be controlled by the college of arts and sciences?

The time has come for a careful survey by educational experts of what is needed for the premedical course. Similarly a survey of what is needed by the general practitioner is required to determine the content and method of the medical curriculum.

THE CHIROPRACTIC BILL FOR RECOGNITION IN SOUTH CAROLINA DEFEATED

A strenuous effort was made this year to secure recognition of chiropractic methods in South Carolina. Dr. D. M. Crosson, Senator from Lexington County, led the fight against the bill. He was ably seconded by Senator W. P. Mason of Oconee. The bill was defeated in the Senate. We wish to pay our respects to South Carolina and the men who see and think straight.

Four of our New England States have capitulated and we have had the unpleasant experience of hearing the House chairman of the Committee on Public Health (himself a physician) express the hope that some way might be found whereby chiropractic treatment might be legally provided for citizens of Massachusetts.

Fortunately we have Drs. Abbott and Holden on this committee, who have stood for conservative treatment of measures of this sort.

If the chiropractors succeed in securing enactment of the bill it will be because the majority of the intelligent people of the State have paid no attention to the menace. Even now a spirited protest by all the physicians of the State together with the assistance of intelligent laymen would give the Legislature an understanding of what the people of Massachusetts really want.

If every doctor would write his Senator and Representative a personal letter or see these men and get just one other voter to do the same the tide would be turned most effectively. The chiropractors have petitions from several thousand persons. If this plan is followed the petitioners in opposition would outnumber the chiropractic advocates two to one.

Cannot the profession emerge from its shell of conservatism in Massachusetts and just once demonstrate a united purpose to save this Commonwealth from the disgrace of opening a back door to the practice of medicine?

CARDIOVASCULAR SURGERY

Surgery on the heart and peripheral vessels is still in its infancy, and little information in regard to this special field has been made available to the general medical public. Surgical interference has been instituted in angina pectoris, chronic valvular disease of the heart, and in Renaud's disease. It is delicate work, demanding long years of training and experimentation, skill and courage.

Some of this work has been done in Boston, and on March 26 at the Boston Medical Library a thorough report will be made by the leaders. The surgical treatment of angina pectoris, of mitral stenosis, and of chronic vaso-constriction will be described and discussed at length.

All members of the medical profession, whether or not they be in the Suffolk District or connected with the Boston Association for the Prevention and Relief of Heart Disease, are urged to attend.

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Legislative Matters

HOUSE BILL 1209

THE hearing on this bill presented by Dr. Charles F. Painter, Chairman of the Committee on Medical Education and Medical Diplomas of the Massachusetts Medical Society, was conducted by the legislative committee on public health, March 12. This bill provided for the elimination of two provisions in the present law: First, that no member of the Board of Registration in Medicine may be a member of the faculty of a medical school, and second, that no medical society shall be represented on the Board by more than three members.

The bill is practically a copy of the present law with these restrictive features omitted and provides for the creation of a new Board or reappointment of the present Board by the Governor. This does not mean a different personnel unless the Governor should so decide.

It was stated by the petitioner that Massachusetts has the lowest requirements for medical registration.

Anticipating the argument in opposition Dr. Painter explained that the poor boy has better opportunities for securing a high-grade medical education today than formerly by reason of the many scholarships and other aids available to deserving students. The class A medical schools of this country have accommodations for 17,000 medical students, which is more than the present enrollment; hence, there is room for all well prepared boys.

He explained that this bill is not meant as an attack on the present Board for it would not prevent the Governor from appointing all of the members on the Board at the present time, but that in future when vacancies may occur the appointments could be made of men who may be especially well qualified to serve but who are now debarred.

One member of the Committee read statements which appeared in a daily newspaper, evidently trying to disconcert Dr. Painter.

Mr. Nicholas Parker, of the Massachusetts Civic League, Dr. F. J. Cotton, of the Boston Chamber of Commerce, Dr. Rowe and Dr. Begg, of the Boston University School of Medicine, Mr. Edward R. Warren, of the Board of Managers, and Dr. John Adams, of the Staff of the Boston Dispensary, Mr. Gordon, of Somerville, Miss M. P. Webster, of the Massachusetts League of Women Voters, and Mrs. F. T. Lord, of the Boston City Federation, spoke in favor of the bill.

The opposition was opened by Dr. Calderwood, the Chairman of the Board of Registration in Medicine, and later he was followed by his associates, Dr. Corr, Dr. Houghton and Dr. Prior. This majority representation of the Board indicates the attitude of this body. Hitherto unofficial expressions of opinion have led many to feel that the sentiment of the Board was definitely in favor of accomplishing all that is sought under this bill, but there seemed to exist in the minds of the members a fear that in the reorganization the personnel would be changed and, although six of the seven members are appointees of the present Governor, doubt may have existed as to the likelihood of reappointment.

The trend toward unsectarianism in medicine has been so pronounced in recent years that it is surprising to find among those who represent the medical societies any of the old fear that any one organization would try to control medical affairs, but the suspicion was clearly expressed and, furthermore, the possibility of favoring the graduates of a medical school by a member of the faculty of the same school was suggested.

This last argument has been applied to present conditions by some who have tried in the past to discredit the work of the Board, for it has been suggested over and over that members affiliated with certain schools, although not on the teaching staff, have discriminated in marking papers.

Since it is believed by most persons competent to form an opinion that the members of the Board are above such practices, it would be at least generous to assume that members of medical school faculties would be fair-minded and reliable.

All of these arguments were taken up by several speakers and the usual references to the medical trust, the dominating influence of the A. M. A. and abuse of the poor boy were repeated.

A State Senator suggested that Dr. Painter, the petitioner, was masquerading under a white robe and hood and the hand of the Ku Klux was to be seen in the bill.

He even allowed his imagination to carry him to the extent of suggesting that the A. M. A. is a subsidiary of the Standard Oil Co., and that this bill must, therefore, be the product of the purpose of Mr. Rockefeller to rob the poor just as the speaker claimed had been the practice of the Standard Oil Co.

He then turned his attention to the medical profession, claiming that doctors did not know the nature of the illnesses of their patients, until told by the patients, and further gave some of his opinions about medicine and disease, claiming that all children should be exposed to scarlet fever in order to have the disease early in life and secure immunity. He then branched off onto the service rendered by medical men in the war, claiming that the representatives of class A schools did not do the actual work but wore the decorations, while the real service was mainly by men from the class C schools. The whole argument was an exhibition of verbal buffoonery.

After several others had argued against the bill a person introduced a report of Dr. Codman's Chicago speech and filed it with the committee. What relation that had to the bill under consideration did not appear. The committee had difficulty at times keeping the arguments of opponents applicable to the subject-matter of the bill.

Most of the speeches were very much like those presented whenever bills to amend the laws relating to medical registration are up for consideration. The unique feature was the union of the Board of Registration in Medicine with the usual opponents of better laws. The Board has not asked for legislation, and it is, therefore, to be inferred that it is satisfied with present conditions.

If this is the attitude of the Board it seems inconsistent because of repeated references to the acknowledged low standards existing in Massachusetts.

If the Board would assume leadership it is very probable that the medical societies would be glad to fall in line and try to avoid working at cross purposes.

Miscellany

COMMITTEES OF THE BOSTON TUBERCULOSIS ASSOCIATION

The President of the Boston Tuberculosis Association, John B. Hawes, 2d, M.D., announces the personnel of the various committees of the association to serve during the ensuing year.

Prendergast Preventorium Committee, which has general management of the camp at Mattapan: George S. Hill, M.D., chairman; Cleaveland Floyd, M.D., Randall Clifford, M.D., John B. Hall, M.D., James J. Minot, M.D., Miss Isabel F. Hyams, Miss Lilian V. Robinson and Miss Julia C. Prendergast.

Prendergast Preventorium Equipment Committee, which is charged with looking after the equipment: Mrs. Frank S. Price, chairman; George S. Hill, M.D., Mrs. William H. Ames, and Mrs. George S. Parker.

Placement Committee, which is undertaking a work that is new to voluntary health agencies in the country: John B. Hawes, 2d, M.D., Randall Clifford, M.D., Harry Linenthal, M.D., Mr. Eliot Farley, J. Danforth Taylor, M. D., Mr. Nathaniel K. Wood, and Miss Mary E. P. Lowney.

Educational Committee, which has the functions also of a publicity committee: Edward O. Otis, M.D., chairman; Gerardo M. Balboni, M.D., Cleaveland Floyd, M.D., Rev. J. P. McCormack, Mrs. George Gilbert Smith, and Mrs. Channing C. Simmons.

Budget Committee: Mr. George S. Mumford, chairman; George S. Hill, M.D., Edward O. Otis, M.D., and Mr. Albert V. Woodworth.

Committee on Legislation: John B. Hawes, 2d, M.D., and Edward O. Otis, M.D.

Committee on Finance: Mr. George S. Mumford, chairman; Mr. Hugh Bancroft, Mr. Horatio Lamb, and Mr. Robert Saltonstall.

A Seal Sale Committee of twelve members was also named, with Cleaveland Floyd, M.D., for chairman. This will become very active next autumn.

BOSTON TUBERCULOSIS ASSOCIATION

The Boston Tuberculosis Association has transferred its stereopticon and slides to the clinics of the Massachusetts Charitable Eye and Ear Infirmary. This is one of the bits of educational work of the association. It seeks to utilize the time that patients must wait with interesting and instructive lantern views. Instead of musing over their troubles, as is usual in clinics where it is necessary to wait to be attended to,—and the clinics in Boston are so popular that they all are in this class,—views of scenery, bright colored birds or pretty flowers are shown, together with helpful texts about health. The series includes the famous "talkless lecture," prepared by the National Tuberculosis Association, views of the happy children at the Prendergast Preventorium of the Boston Tuberculosis Association, or pictures of the places that the State furnishes for the benefit of those who have lost their health. The views have been shown at Boston Dispensary the past few months, and have attracted much attention and comment. The lantern is operated by Mrs. Edward Heffernan, of 25 Fenno Street, who is the leader of a group of ladies of the Roxburghe Club, interested in helping their fellow citizens.

SUMMER COURSES IN PUBLIC HEALTH SCHOOLS

The United States Public Health Service has announced that, in response to an extensive demand for summer school work in public health, it has arranged with Columbia University, the

University of California, the University of Michigan, and the University of Iowa to conduct public health summer schools this year.

The faculties of these various summer schools will include many such leading specialists of the United States as Michael M. Davis (dispensary management), Robert H. Gault (criminal psychiatry), Emery Hayhurst (industrial hygiene), William J. Mayo (non-communicable diseases), E. V. McCollum and H. C. Sherman (nutrition), William H. Park (laboratory methods), Earl B. Phelps and George C. Whipple (public health engineering), M. J. Rosenau and Victor C. Vaughan (epidemiology), Thomas W. Salmon (psychotherapy), John H. Stokes (syphilis), Philip Van Ingen (child hygiene), C.-E. A. Winslow (public health administration), and Francis Carter Wood (cancer).

The Public Health Service has already received communications from several thousand physicians and sanitarians who hope to attend these summer schools. The widespread interest manifested thus early indicates that a large number will take advantage of this opportunity.

Further information may be obtained by applying to

H. S. Cumming, *Surgeon-General.*

Obituary

PERLEY PIERCE COMEY, M.D.

Word has been received of the death in Augusta, Ga., on March 10, 1924, of Dr. Perley Pierce Comey, at the age of seventy-two.

Dr. Comey was a native of Holliston, Mass., where he was born, January 14, 1852. After graduating from Harvard Medical School, in 1878, he settled in practice in Clinton where he remained until 1897, moving to Worcester in that year. In 1910 he took up his residence in Augusta and there became associate professor of medicine in the University of Georgia.

He was a member of the Massachusetts Medical Society after 1879, his name being placed on the retired list in 1920; also of the Medical Association of Georgia, and the American Medical Association.

He is survived by his widow, a son and a daughter.

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For Women: "Get your red lips in the grocery store—not in the drug store. Blood deficient in hemoglobin or red coloring matter, indicating a mild degree of anemia, is a very common finding in periodic health examinations. When a large group of young people—school children, clerks, or workers in industry—are examined, a low hemoglobin reading is so common as to be accepted almost as a matter of course; that is, as a characteristic of civilized life. A low hemoglobin among young women is particularly notable."—*Life Extension Institute.*

The Massachusetts Medical Society

District Secretaries are requested to compare their lists of members with the list of changes from January 1 to March 1, 1924, published in the Journal of March 13, pages 467, 468, 469, and to notify the Secretary of the Society of any errors noted.

The Massachusetts Medical Society is on a little higher plane than some of the State medical societies because it spends time and effort in truing up its constantly changing membership. Let us have coöperation.

News Items

Removal.—Dr. Frank S. Cruickshank has removed his office from 53 Willow Street, West Roxbury, to 520 Beacon Street, Boston.

L. Emmett Holt, M.D., LL.D., 1855-1924.—A special meeting was held in commemoration of the life and services of Dr. Holt, at the New York Academy of Medicine, 17 West 43rd Street, Wednesday, March 12, at 8:30 p.m.

Beverly Hospital.—A demonstration clinical meeting was held at the Beverly Hospital, Tuesday, March 18, at 4 p. m. Interesting cases were shown and discussion followed.

Lawrence General Hospital.—A clinical staff meeting of the hospital was held on Tuesday, March 11. A Series of Hips was the subject of the evening, and B. E. Wood, M.D., of Boston, was the chairman.

The Franklin District Society.—The regular meeting of the Franklin District Medical Society was held at the Weldon, Greenfield, on Tuesday, March 11, at 11 a. m. Program: "Diagnosis of Surgical Conditions of the Kidney and Ureter," Dr. W. O. Wilder, Springfield, Mass. This paper was illustrated with lantern slides.

Correspondence

COUNCIL ON PHARMACY AND CHEMISTRY OF THE A. M. A.

The following named articles have been accepted:

Abbott Laboratories: Butesin Picrate; Butesin Picrate Ointment.

Parke, Davis & Co.: Dibromin.

E. L. Patch Company: Patch's Flavored Cod Liver Oil.

Vitalait Laboratory of California: Vitalait Culture Bacillus Acidophilus.

Wilson Laboratories: Epinephrin-Wilson; Epinephrin Powder-Wilson; Epinephrin Solution, 1:1000-Wilson.

W. A. Puckner, *Secretary.*

THE BANTING RESEARCH FOUNDATION

TORONTO, CANADA

March 6, 1924.

Mr. Editor:

Sir—The discovery and development of Insulin by Dr. F. G. Banting, Mr. C. H. Best and other coöperating investigators has brought relief to a multitude of sufferers from diabetes throughout the world. At a low price this boon has been placed within reach of all. But it is well known that only a beginning has been made in alleviation even of this one malady. Notwithstanding the magnificent advances that have been effected in arresting or averting many of the most grievous attacks of disease on human life, mankind is beset by enemies. Their strategy must be discovered and circumvented. This can be done only by patient research conducted in the main by skilled investigators who devote their lives to scientific inquiry. For these investigators the public at large must provide the means of support, for they it is who benefit immensely thereby. Such work has been going on quietly all over the world. Laboratories in the universities have groups of investigators working in coöperation under the direction of competent scientists. But only now and then does a result such as Dr. Banting achieved strike the imagination of the world. It is therefore but appropriate that advantage should be taken of it to appeal to the grateful public for support in making possible the continuance and prosecution of this work and of other investigations in medical science. To effect this and to signalize the discovery and the development of Insulin, the Banting Research Foundation has been created.

The purposes of this Foundation have been defined to be:

(a) To provide, in the first instance, further funds for the support of the Banting and Best Chair of Medical Research at the University of Toronto.

(b) To establish a fund for the adequate financial support of such scientific workers as may have proposed definite problems of medical research, and for whom funds are not otherwise available. Such assistance may be given to persons working in the University of Toronto or elsewhere.

All financial arrangements in connection with the collection and reception of the principal and subsequent expenditure of the income of the fund have been vested in a board of trustees, the members of which are appointed for a term of three years, subject to reappointment at the end of their respective terms of office. Trustees have now been appointed as follows:

Sir Robert A. Falconer, K.C.M.G., D.Litt., LL.D., D.D. (Edin.), D.C.L. (Oxon.), president of the University of Toronto, chairman.

Lieutenant-Colonel R. W. Leonard, member of the Board of Governors of the University of Toronto, honorary treasurer.

The Rev. Canon H. J. Cody, D.D., LL.D., chairman, Board of Governors, University of Toronto.

C. S. Macdonald, Esq., M.A., general manager, Confederation Life Association.

W. E. Gallie, M.D., F.A.C.S., F.R.C.S. (Eng.), surgeon-in-chief, Hospital for Sick Children, Toronto.

Professor J. G. FitzGerald, M.D., F.R.S.C., professor of hygiene and preventive medicine, director, Connaught Laboratories, University of Toronto.

Professor V. E. Henderson, M.A., M.B., professor of pharmacology, University of Toronto.

Mr. John W. Rogers.

The trustees propose to make an appeal to the public for funds in the immediate future. In the meantime they desire to bring these facts to your attention and they hope that you will be good enough to communicate them to your readers.

Believe me,

Yours faithfully,
F. Lorne Hutchison,
Honorary Secretary.

P. S.—Subscriptions to the fund will be welcome at any time and should be made payable to the Banting Research Foundation, Toronto, Canada.

VIENNA LETTER

Vienna, 28 January, 1924.

The Cost of Living in Vienna.—Food and other prices and the general cost of living has lately been one of the matters before Parliament. There is a general consensus of opinion that prices are still too high in Vienna, quite apart from the tendency to increase, which has of late begun to manifest itself unpleasantly here, and a determined effort is to be made at headquarters to bring about a continuance of the downward course which prevailed from the spring of 1923 to the autumn in 1923. During that period, thanks mainly to the improvement and consolidation of the Austrian currency, very considerable reductions took place. Wholesale food prices fell by 25 per cent., those of other commodities by nearly 40 per cent. Retail prices of foodstuffs sunk also considerably; those of other commodities by about one third. The Minister of Food gives the average cost of living for a working class family of five persons as having fallen from a point represented in March by the figure 13,650 to a point represented in November by the figure 11,080. The government is now determined to see that the ground thus won is not going to be lost, and that the recent remission of taxation and reduction of coal prices shall have its due effect in bringing down the general cost of living to a still lower level.

The Housing Problem.—Since the war the housing question has been one of the most acute problems in Austria as elsewhere. Here it is the more keenly felt as an enormous quantity of emigrants from the successor states of the Late Austria-Hungary took refuge and settled. Of course, this fact has caused that even the formerly uninhabitated dwellings are being now occupied by families. These dwellings being unhygienic, form the hotbed of infectious and contagious diseases. The enormous increase in the price of building materials, owing to the depreciation of the currency and the measures taken to protect tenants from being exploited by the owners who would otherwise have raised the rents to an excessive degree,—all this helped to weaken the financial resources of the building movement which was necessary, especially in Vienna. In order to ease the situation the State decided to take the initiative, at any rate temporarily, and through the Ministry of Social Welfare supported the construction chiefly of family flats, etc. It was a question not of building tastefully, but of building at all costs. According to the building law of 1920 the State made a building grant of a huge sum, for the construction of houses, the greater part of which is already completed. They consist in the main of houses built by coöperative societies and municipalities. The State contemplates altering the existing law, arguing that the economical condition of the State is consolidated and the prices went down considerably and therefore the State grant can be correspondingly reduced. Hence, the State subsidy under the new law will cover 45-50 per cent. of the building outlay instead of 70 per cent. as hitherto. This proves that the government only intends the building subsidy to be an emergency measure which will be withdrawn as soon as economic conditions allow it.

Insulin.—Insulin is being employed already in all clinics, hospitals, and ambulatories of Austria. The price of foreign-made insulin was so prohibitive that only the well-to-do classes could avail themselves of its great blessing in severe diabetic cases, up to the summer of last year when the German preparations made their appearance, which were sold well under the price asked for English and American insulin. Despite this fact many eminent physicians preferred to use the English insulin, having no reliance in other makes. Of course, their distrust has faded away with the time, because the experiments made in the different clinics with German-made insulin have proved, undoubtedly, their equality with the foreign preparations. Insulin is being manufactured now in Austria, too, and it is sold at a considerably lower price than obtains in America or England, although it is said to be made in these countries only with a view to public utility and, therefore, sold at cost price. English insulin, until lately, was seventeen shillings and sixpence wholesale (cost price), whereas the Austrian preparation is sold at 150,000 crowns (about three dollars) per 100 units, wholesale, inclusive of manufacturers' profits. Trade papers suggest the likelihood of insulin made in this country finding an export market abroad.

Factories and Health.—In the Vienna Medical Society Dr. Wenkebach, speaking about the present state of the health in the industrial districts of Austria, said that it is generally accepted as a matter of course that the introduction of factories and workshops into any district where they have not previously existed results in the deterioration of the health of the population. That this is not always so—and that it need not be so—he pointed out with the case of a small town, showing that the industrializing of country districts is carried out with due regard to the welfare of the workers. This may not only result in greater prosperity, but in a higher standard of living and better health. Twenty-five years ago, before industries were started in the Steyr district, the food of the people was of a poor nature, and the number of deaths among the population was in the ratio of 1:58, 5 per cent. of the mortalities having been due to tuberculosis. The town of Steyr has now one of the largest automobile and cycle and arms manufactories in the country, workmen dwellings in up-to-date style have been erected by the factories, and the standard of living in the district is equivalent to that of a large town. Last year the mortality rate was only 1:82. There is no reason why such cases should not be the rule and not the exception, says Dr. Wenkebach.

The Activity of the Czechoslovakian Red Cross Society.—The Czechoslovakian Red Cross Society, which can now look back upon five years of activity, raised during that period the sum of 179,634,936 crowns, the equivalent of considerably over five million dollars. Of this sum, 119 million crowns have been spent in relief and provision for health and hygiene. The Red Cross First Aid corps has been recruited by 15,411 members from the various fire brigades throughout the country. The Junior Red Cross, recruited mostly from the schools, has a membership of approaching 200,000, and is numerically the second largest Junior Red Cross Society of the world, ranking next after that of the United States of America. In September and October last the Junior Red Cross contributed 40,000 of the 475,000 crowns raised by the parent society and the Legionaries' Union for the relief of the sufferers from the earthquake in Japan.

Numerus Clausus in the Czechoslovakian Universities.—A leading member of the German Nationalist party spoke this week in the Prague Chamber of Deputies in favor of applying the *numerus clausus* in the universities, and other higher educational institutions. Under the numerus clausus the number of Jewish students allowed to study at the universities, etc., and the number of professors of Jewish persuasion allowed to teach would have to be proportional to the total Jewish population of the country. Any excess would be prohibited. This idea of denying public higher education, or the rewards of it to a certain class of the population, merely on account of their religious belief, is, of course, a common phase of Pan-German mentality throughout all Central Europe, manifesting itself particularly in anti-Semitic demonstrations in Budapest, and also in Vienna. It finds no toleration among Czechoslovaks and even in the German parliamentary minority of 72 members in the Prague Chamber of Deputies, only 15, supported by five Hungarians, could be found to back the proposal. The Germans in Czechoslovakia owe a large degree of their cultural level to the Jews, but the Pan-German section do not evidently feel in debt.

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ACKNOWLEDGMENT OF BOOKS FOR REVIEW

The Journal acknowledges the receipt of the following books for review:

An Introduction to Surgical Urology. William Knox Irwin. New York: William Wood & Co. Pp. 180. Price $2.50.

Marriage and Syphilis. Dr. George Katsainos. Boston: Wright & Potter. Price $3.00.

Hernia. Leigh F. Watson, M.D. St. Louis: C. V. Mosby Co. Pp. 660. Price $11.00.

Practical Chemical Analysis of Blood. Victor C. Myers, Ph.D. (Second edition.) St. Louis: C. V. Mosby Co. Pp. 232. Price $4.50.

Management of the Sick Infant. Langley Porter and William E. Carter. St. Louis: C. V. Mosby Co. (Second edition.) Pp. 659. Price $8.50.

Geriatrics. Malford W. Thewlis. St. Louis: C. V. Mosby Co. Pp. 401. Price $4.50.

Fighting Foes Too Small to See. Joseph McFarland. Philadelphia: F. A. Davis Co. Pp. 309. Price $2.50.

Intranasal Surgery. Fred J. Pratt and John A. Pratt. Philadelphia: F. A. Davis Co. Pp. 334. Price $5.00.

Intravenous Therapy. W. F. Dutton. Philadelphia: F. A. Davis Co. Pp. 542. Price $5.50.

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NOTICES

By advice of a majority of the members of the committee in charge of the Journal and the editorial staff, the custom of furnishing a number of reprints free to authors is discontinued.

The expense of the free reprint custom has put a considerable burden on the Society.

Reprints will be furnished at cost and every effort will be made to accommodate authors.

The American Climatological and Clinical Association will meet at The Ambassador, Atlantic City, for its annual convention, May 1-2-3. One hundred and fifty doctors are expected to attend.

The American Urological Association has also completed arrangements to return to The Ambassador, where it met two years ago. This association will spend two days at the shore, June 3 and 4.

THE NORFOLK DISTRICT MEDICAL SOCIETY

A regular meeting of the society will be held at Masonic Temple, 171 Warren St., Roxbury, April 1, at 8.15 p. m.

Business:

Communication:

Bleeding in the Last Half of Pregnancy, Robert L. DeNormandie, M.D.

Laboratory Aids in the Toxemias of Pregnancy, Judson Smith, M.D.

Treatment of Toxemias of Pregnancy, James R. Torbert, M.D.

Refreshments after the meeting.

BRADFORD KENT, M.D., *Secretary.*

HARVARD MEDICAL SOCIETY

Announcement of the meeting, Tuesday, March 25. Topic: Bronchoscopic and Esophagoscopic Cases of General Medical and Surgical Interest. Lantern and moving picture demonstration by Dr. Chevalier Jackson, Dr. Gabriel Tucker and Dr. Louis H. Clerf of Philadelphia at 8.15 in the Amphitheatre of Building C, Harvard Medical School, instead of at the Peter Bent Brigham Hospital.

HARVEY CUSHING, *President,*
GILBERT HORRAX, *Secretary.*

DISEASES REPORTED TO DEPARTMENT OF PUBLIC HEALTH

WEEK ENDING MARCH 1, 1924

Disease	*No. of Cases*	*Disease*	*No. of Cases*
Anterior poliomyelitis	2	Ophthalmia neonatorum	18
Chicken-pox	309	Pneumonia, lobar	168
Diphtheria	205	Scarlet fever	527
Dog-bite requiring anti-rabic treatment	12	Septic sore throat	7
Encephalitis lethargica	1	Syphilis	38
Epidemic cerebrospinal meningitis	5	Tetanus	2
German measles	27	Suppurative conjunctivitis	39
Gonorrhea	99	Trachoma	4
Influenza	8	Trichinosis	2
Measles	926	Tuberculosis, pulmonary	100
Mumps	467	Tuberculosis, other forms	13
		Typhoid fever	7
		Whooping-cough	109

PUBLIC HEALTH LECTURERS FOR THE YEAR 1924

The Committee on Public Health of the Massachusetts Medical Society has been able during the past few years to arrange with well known specialists in various medical fields to give talks at meetings of the District Medical Societies on subjects of interest and importance to all practitioners. It is a pleasure to announce that a similar arrangement has been made this year and that the gentlemen named below are willing, without expense to the District Society, to give occasional talks of thirty to forty minutes on subjects relating to the promotion of public health, extending opportunity for questions and discussion. It is suggested that medical societies consider meeting at neighboring public institutions, since such meetings have been most successful in the past, particularly at the tuberculosis sanatoria and state hospitals for the insane.

José Penteado Bill, M.D., Doctor of Public Health. Specialty: Preventive Medicine.

Frank C. Dunbar, M.D., Bacteriologist, Instructor in Bacteriology and Pathology, Tufts College Medical School. "Methods of Technique in Collecting Specimens."

Walter E. Fernald, M.D., Superintendent, Massachusetts School for the Feeble-minded.

Timothy Leary, M.D., Professor of Pathology, Tufts College Medical School; Medical Examiner, Suffolk County.

Herman A. Osgood, M.D., X-Ray Department of the Boston City Hospital: "Focal Infection."

Edwin H. Place, M.D., Physician-in-Chief, South Department, Boston City Hospital. Specialty: Contagious Diseases.

C. Morton Smith, M.D., Chief of Department of Syphilis, Massachusetts General Hospital.

George Gilbert Smith, M.D., Assistant in Department of Genito-Urinary Diseases, Massachusetts General Hospital. Specialty: Genito-Urinary Diseases.

Lesley H. Spooner, M.D., on Staff of Boston Dispensary. Specialty: Specific Diagnosis and Treatment of Pneumonia.

George H. Wright, D.M.D., Lecturer on Dental Hygiene, Harvard Dental School. Specialty: Dental Surgery.

Thomas F. Kenney, M.D., Director Public Health and Hygiene, City of Worcester. Specialty: Full-Time School Health Officer.

Paul Dudley White, M.D., Chief of Medical Out-Patient Department and of the Cardiac Clinics, Massachusetts General Hospital. Specialty: Cardiovascular Diseases.

Gilman Osgood, M.D.: "A General Practitioner's Observations on the Relation of Oral Infection to Disease."

Francis H. McCrudden, M.D., Professor of Applied Therapeutics and Internal Medicine. Specialty: Health Examinations.

Secretaries of District Medical Societies, writing to ask for these lecturers, will kindly designate the topic, the place and the hour of meeting as well as the name of the desired speaker, thus eliminating unnecessary correspondence. Please address communications to the Secretary of the Committee, Annie Lee Hamilton, M.D., 164 Longwood Avenue, Boston 17.

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SOCIETY MEETINGS

DISTRICT SOCIETIES

Bristol South District Medical Society:

The annual meeting will be held in New Bedford, May 1, 1924.

Essex North:—Annual meeting at Lawrence General Hospital, May 7, 1924.

Essex South District Medical Society:

May 7, 1924:—Annual meeting, Relay House, Nahant, in conjunction with Lynn Medical Fraternity.

Franklin District:—Society meets at Greenfield the second Tuesday of March, May, July, September. Annual meeting in May.

Hampden District:—The meetings for the year are as follows:

April, 1924, at Springfield; annual meeting.

Hampshire District Medical Society:

Meetings held bi-monthly, the second Wednesday in the month, at Boyden's Restaurant, Northampton.

Middlesex South District Medical Society:

March, 1924:—Hospital meeting; place not yet determined.
April, 1924:—Annual meeting.

Norfolk South District:—Meetings first Thursday of each month at 11.30 a. m., April and May, at United States Hotel, Boston. The May meeting is a stated meeting.

Suffolk District Medical Society:

March 26, 1924:—Meeting of the Medical Section, in association with the Boston Association for the Prevention and Relief of Heart Disease, at the Boston Medical Library at 8.15 p. m.
April 30, 1924:—Annual meeting to be held at the Boston Medical Library at 8.15 p. m.

Worcester District:—The meetings for the year are as follows:

April 10—A public meeting.
May 8—Annual meeting.

STATE, INTERSTATE AND NATIONAL SOCIETIES

Schedule of meetings of the *New England Dermatological Society:*

Wednesday, April 9, 1924, at 3 p. m., in the Surgical Amphitheatre, Boston City Hospital.